AN INTRODUCTION TO CARDIOLOGY

BY

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PREFACE

THERE are two ways of acquiring knowledge Known facts can be stored in the memory, or the general principles which govern the relation of facts to one another can be learned Only an unusual mind can store a mass of isolated detail, but if underlying general laws and principles are understood, these are remembered and can be used to clarify new situations as they arise This is the soundest approach to clinical medicine, for every case is different and the variation in the problems set can never be covered by an arbitrary series of rules

The chief function of good teaching is to portray the structure of a subject so vividly that the relations and shape of the composition are clear and balanced It is not without reason that teachers are remembered by their personalities, for as an artist depends upon his personal sense of pictorial, literary, or musical values so does a teacher rely upon his power of giving shape to his subject To achieve this, light and shade must be contrasted, some details emphasised and others omitted

So should it be with books On the one hand there is the textbook, which is essential as a storehouse of information, but the more detailed and complete it is in its accumulation of facts the less becomes its accessibility as a means of acquiring the form or shape of the subject Therefore there would seem to be a place for books, more readable in type if less full of stored knowledge, whose function should be to stimulate interest and to inculcate a sense of shape form, and general principles

The purpose of this book is to portray, in successive chapters, the chief aspects of cardiovascular disease The matter is largely derived from lectures given at St Bartholomew's Hospital, and from clinical teaching over cases, and is influenced by these aspects of cardiology It is realised that such a method entails the omission of detailed and minute information But an attempt has been made to stress the essential underlying laws and tendencies which mould clinical opinion It is hoped that this mode of presentation may provide both an interest in cardiology and a sound scaffold for the erection of a fuller knowledge

It is clear that a decision has of necessity been taken as to the

scope of the book the limit has been set in the various subjects with reference to the supposed powers of clinical judgement of the reader in relation to the cases he is likely to meet Few general practitioners or well equipped hou e officers see a sufficient number of unusual clinical cases rare cardiograms or atypical radiograms, to render their judgement completely reliable in such matters especially when a patient s life or future health is at stake, but most of such men and women see a sufficient number of cases of less rarity to serve as a basis upon which the soundness of their clinical judgement can be progressively increased If here and there the set limit has been overstepped the reason is that the author has been carried away by some matter of particular interest to himself This is a fault in all teachers which is inseparable from their human attributes The formed specialist will consult the literature and the textbook for his factual information

Books on heart disease are difficult to shape The subject may be treated from the different aspects of physiology anatomy pathology physics and chemistry A case of mitral stenosis for example may well present one or more of the following problems acute rheumatism as a cause of myocardial change failure of the left side of the heart with pulmonary congestion the interpretation of the thrills and murmurs of valvular disease cardiac irregularity with its treatment the pharmacology of digitalis, quinidine and the diuretics radiology and finally electrocardiography

The plan of the present book has been devised on the basis that failure of the heart s function is the central cardiological problem facing the physician Therefore after an introductory section dealing with methods of examination the subject starts with a description of heart failure and its effect upon the body This is followed in what is hoped to be a logical sequence by a description of various factors which may produce heart failure First there are those which injure the strength of the heart muscle Then in succession there come the various handicaps to proper cardiac action to begin with the abnormalities of function such as the irregularities then the mechanical deterrents to good cardiac action pericarditis endocarditis congenital malformation and finally the *influence of arterial disease* This latter is subdivided into the lesions of coronary arteries with cardiac pain and the effects of hypertension

No attempt has been made at providing a comprehensive bibliography for the average busy practitioner and senior student will be unlikely to utilise one The reader is especially referred to *Heart*

Disease, by Paul D White (Macmillan, 1944), as the best detailed textbook on the subject, also to *Electrocardiography in Practice*, by Graybiel, White, Wheeler and Williams (W B Saunders Company, 1946), and to *The Clinical Roentgenology of the Cardiovascular System*, by H Roesler (Baillière, Tindall and Cox, 1938)

Since it has been the writer's custom to rely on X-ray screening and not to take his own X-ray films, he is much indebted to Dr George Simon for help in selecting an appropriate series of radiographs as illustrations He is also glad to record his appreciation of the valuable help given by Dr Simon in many cases of radiological difficulty Finally he wishes to thank the editors of the *Lancet*, *British Medical Journal*, the *Practitioner*, and the *North Eastern Counties Medical Journal* for their kind permission to allow the republication in this book of the following papers published by them Some necessary modifications and abbreviations have been made in this process The papers concerned are

"Cardiac Signs in Symptomless Young Adults" Published in the *Lancet*, 30th Nov 1946, p 779

"Treatment of Rheumatic Heart Disease" Published in the *Practitioner*, Dec 1941, vol 147, p 737

"Treatment of Syphilitic Heart Disease" Published in the *Practitioner*, May 1937, vol 138, p 585

"The Heart in Thyrotoxicosis" Published in the *British Medical Journal*, 1935, vol 1, p 1277

"Cardiac Symptoms and Signs in General Medical Diseases" Published in the *Newcastle and North Eastern Counties Medical Journal*, Dec 1947

The author in conclusion would like to express his indebtedness to his former chief, Dr J H Drysdale, from whose honest mind and dry humour he first acquired a true appreciation of the critical sense as applied to medicine, to Sir John Parkinson, whose friendly sympathy and whose academic example has for so long been a source of strength to his London colleagues, and to Dr Paul D White, whose visits to England, too infrequent though they have been, always bring such a rich cargo of scientific stimulation

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Section I

EXAMINATION OF THE HEART

CHAPTER I

HISTORY-TAKING AND PHYSICAL EXAMINATION

THE purpose of an examination of the heart is to determine whether the function of the organ is normal, or is in any way diminished, also to decide from the available evidence whether there is any possibility of future improvement or deterioration in this function The essential point of the problem is to decide whether the heart muscle is healthy, or whether it is handicapped by any factor which could influence its health either acting directly upon the muscle, or indirectly as in a disturbance of the valve or the rhythm

The clinical examination can be usefully divided into three stages the history, the physical examination, and the special examination by electrocardiography, X ray, and other subsidiary pathological methods The most important of these three is the history, and it can never be stressed too strongly that *the taking of a really adequate history, with clear statements upon details, constitutes 50 per cent. of the evidence which may be available in nearly every case.*

The two qualities most necessary for taking a history are reasonable and persistent scepticism and an insatiable curiosity Every statement of the patient should be listened to carefully, and verified by kindly cross examination Leading questions, so far as is possible, should be avoided Every statement the patient makes however apparently irrelevant, should be treated with respect and carefully noted

It is helpful when taking a history to have a printed form divided into sections for the past history, family history, and history of the present condition The specimen shown overleaf is an example

When the patient's own symptoms have been fully transcribed in chronological order, and when every possible fact seems to have been extracted from the patient's story, additional enquiry is then made with regard to those other symptoms to which reference has not been made In this way a fairly full account is obtained which is unlikely to have missed many important points

Special attention should be paid to the section marked "Activities"

The character of the patient's work should be minutely investigated, with especial stress upon the length of hours, the physical or mental burden, the time set aside for meals, and the general effect of these upon the symptoms already complained of. Information about the length and difficulty of the journey to and from work must also be elicited. Further details with regard to the history vary enormously according to the particular type of illness from which the patient

Past History			*Family History*
Rheumatic fever	Scarlet fever	Syphilis	
Growing pains	Tonsillitis	Other diseases	
Chorea	Diphtheria	Puerperium	

Symptoms		
Dyspnœa	*Palpitation*	*Bowels*
Sighing	Exhaustion	Catamenia
Orthopnœa	Cough	Frequency
Pain	Giddiness	Œdema
Constriction	*Dyspepsia*	

Activities		
Work	Stairs	Sleep
Games	Running	Habits
Walking		

may be suffering, so that the rest of the pages of this book are only part of the available symptomatic evidence. To take a really full and perfect history postulates a complete knowledge of medicine past, present, and future. Thus although a perfect history can never be taken, *the greater the knowledge of the subject the fuller and* more valuable will the history be.

Physical Examination

The physical examination of the patient is a smaller problem than taking the history, and the principles involved are the same irrespective of the disorder or disease. The old established routine —inspection, palpation, percussion, and auscultation—is the best for all good physical examinations.

The examination room must be quiet and light. It is probably an advantage if the light is oblique rather than coming from above, since slight fullness of veins and slight pulsations are more easily made visible in good light by the presence of contrasting shadow. Circumstances which frighten the patient should be absent, and the general attitude of the examiner and the general surroundings should

be reassuring As with the history, so in the physical examination it is helpful to have a printed list which will enable an exact routine to be followed The specimen which follows is the one used by the writer For the purpose of saving space the sheet has been slightly compressed, but without omission General inspection will reveal the presence of shortness of breath, orthopnœa cyanosis or pallor, pulsation, clubbing and tremor If sighing is present it is apt to occur during the taking of the history

Present Condition

Dyspnœa	Pulsation
Orthopnœa	Clubbing
Cyanosis	Tremor
Pallor	
Pulse Rate B P	Rhythm
Arteries	
Veins	Retinæ

Heart

Inspection
Apex beat
Thrills
Percussion
Sounds
Hyperæsthesia

Lungs
Abdomen
Œdema
Urine

Cyanosis may be general or local An easy distinction between local and general cyanosis can be made by submerging the cyanosed hand in warm water for five minutes If the cyanosis is general the colour will remain unchanged if it is local the blue will give place to red or pink

Pallor is a misleading symptom Many pale people are not anæmic and many anæmic people are surprisingly without pallor even in the absence of cosmetics The colour of the mucous membranes of the mouth and conjunctiva are the best clinical guide to the presence of anæmia A good hæmoglobinometer is an essential instrument for the examination of cardiac patients

Pulsation may be thoracic or peripheral *Aneurysms may cause* a general pulsation of the chest, which is often slight and for the determination of which a good oblique light is very helpful Similarly *the slight pulsation inwards constituting Broadbent s sign is best* seen in a good oblique light with the pulsating area not quite in shadow The systolic movements of the chest at and around the apex beat and in the epigastrium must also be noted Pulsation in

the neck may be venous or arterial Venous pulsation is better seen than felt, and arterial pulsation is as easily felt as seen

Tremor must be carefully noted, especially in cases of suspected thyrotoxicosis Clubbing of the fingers may indicate congenital heart disease, chronic pulmonary disease, usually with fibrosis, subacute bacterial endocarditis, or aneurysm of the aorta, in which latter case it may be unilateral

It is upon the heart rate, rather than the pulse rate, that attention must be focused in cardiac patients The reason is that in many irregularities the heart beat fails to reach the wrist The counting of the heart rate is a simple matter, even when this is said to be uncountable from extreme rapidity, provided the rhythm is regular With a stethoscope on the apex, and an eye on the minute hand of a watch, it is nearly always possible to count every fourth beat instead of every beat, and in this way to estimate the rate over half a minute The heart rate will then be twice this multiplied by four The regular heart rate of between 200 and 300 can thus be reasonably well enumerated

If the heart is irregular it is necessary to determine the effect of exertion upon the irregularity In auricular fibrillation the irregularity becomes increased by exercise, whereas in most other irregularities of rhythm the beat becomes more regular with exercise The effect of respiration upon the irregularity is also of importance

Estimation of the blood pressure must be carefully made and should follow a definite routine The cuff is applied firmly, not loosely, to the arm just above the antecubital fossa, and it is helpful so to arrange the cuff that the rubber tubes come off from the border of the cuff remote from the antecubital fossa This area is thus free for the finger and the stethoscope, and the arrangement makes it easier to place the sphygmomanometer behind the patient's head and out of his sight The blood pressure should be taken a number of times, the first estimation, and even the second, will frequently show an increase due to nervous factors A frightened patient should be reassured that there will be no pain, but only a sense of fullness in the arm The systolic blood pressure should always be determined by palpation first, either of the brachial artery or of the radial If this is done mistakes due to the "silent gap" will be avoided In some patients, especially in the presence of hypertension, as the pressure falls from the systolic point, a range of pressures is reached in which the stethoscope picks up no sound from the brachial artery For example, if the systolic pressure is 270,

there may be a silent area between 200 and 160 Unless the systolic blood pressure is taken first by palpation it would be possible to place the stethoscope on the brachial artery in such a patient at 180, when nothing would be heard As the pressure fell to 160 the sounds would appear, so that by auscultation it might be imagined that the systolic pressure was 160, instead of being 270

In taking the blood pressure the diastolic reading is even more important than the systolic The diastolic point is determined as follows when the pressure in the cuff is increased above the systolic point and is allowed to fall slowly, the first sound to be heard is a sharp sound which gives place, as the pressure subsides, to a more muffled sound This in its turn emerges into a second sharp sound, which ends at the diastolic blood pressure figure This second sharp sound is followed frequently for a few millimetres of mercury by a second muffled sound, but this second muffled sound should be disregarded The mercury sphygmomanometer is by far the best instrument

Examination of the thorax and of the heart and lungs is planned along the routine lines of inspection palpation, percussion and auscultation Inspection of the chest will reveal the general shape the movements, respiratory and cardiac, and the amount of expansion during inspiration The straightness or extent of curvature of the spine is also important, as also is the depth of the chest from sternum to vertebral column If the chest is asymmetrical, the heart may well be displaced by this means alone to one side or to the other This may give a false impression of enlargement of the heart The respiratory excursion should be measured by a tape measure Emphysema is an important cause of heart failure

The most important single fact which can be determined by physical examination is the position of the apex beat, for this gives the best clinical information as to the size of the heart *The heart which is enlarged is, or has been, diseased The heart which is not enlarged is most unlikely to be diseased* The 'apex beat" is the arbitrary term given to that position on the chest surface, farthest to the left and farthest downwards, at which the heart's impulse can be definitely detected by the finger in a soft interspace *The apex beat is not the point of maximum impulse* In many patients the apex beat can be easily defined, but in some this is difficult and even impossible, for instance both emphysema and increased thickness of the chest wall, where this increase is due to adipose tissue or to other cause, can interfere with the clinical localisation of the cardiac impulse and therefore with that of the apex beat

Having determined the position of the apex beat, its distance from the mid line is measured. It is obvious that there is a great variation in the size of the normal thorax, in children of different ages, and in adults of varying builds. The normal figure, in inches or centimetres, will differ correspondingly with the individual case. For this reason the mid clavicular line is used as a standard to determine whether the apex beat is normal in position or displaced. The nipple line is clearly not constant in position in the female sex. The mid-clavicular line is the better standard of comparison, and it must be determined with accuracy. The normal apex beat should be inside the mid-clavicular line.

In order to measure the position of the apex beat, in its relation to the mid-clavicular line, the following routine is used. The sterno-clavicular joint is marked with a skin-pencil, and so is the claviculo acromial joint. Thus the length of the clavicle is accurately determined in inches or centimetres. This is bisected and the central point, so determined, is also marked. The distance between this mid clavicular point and the centre of the neck is then defined and measured. The apex-beat measurement can then be compared accurately with that of the mid-clavicular point in inches or centimetres. In some patients in whom the heart is beating vigorously, or in whom left ventricular hypertrophy is present, the cardiac impulse may thrust strongly at the ribs, and if the hand is placed over the ribs it may receive the wrong impression as to the actual position of the apex beat. In such cases careful palpation with the finger-tips in the interspace is necessary.

Thrills, systolic, diastolic, or presystolic, are best felt with the flat of the hand pressed firmly against the chest. Some individuals feel a thrill most easily with the palm of the hand and others with the proximal part of the fingers. In both cases firm pressure of the examining hand is very necessary. The ease with which thrills are felt also varies with the position of the patient, some thrills being felt best with the patient upright, others with the patient lying flat, and mitral thrills with the patient in the left lateral position. A thrill is always accompanied by a corresponding murmur, but the relative *intensity of the two varies, so that whereas in some cases the murmur* may be loud and the thrill slight, in others the thrill is easily felt but the murmur is distant.

Percussion is a method of examination which has been abandoned by some and retained by others. There is no doubt that some examiners can obtain useful information from the measure, whereas others fail. The difference is probably determined by the possession of, or

the lack of, a sense of musical pitch If the examiner is devoid of this, he is unable to detect the change of note which is quite clear to the individual who has a sense of pitch Percussion must be done with the plessor finger firmly applied to the chest wall, and lying parallel with the ribs

The use of the stethoscope needs also some discussion There are two main varieties of the instrument those of the bell type, and those with a diaphragm The diaphragm is most useful for picking out sounds and murmurs of a high frequency, such as an aortic diastolic murmur The bell mouthpiece is better for picking up the lower-pitched mitral murmurs The stethoscope in which both of these are combined is thus valuable Some individuals find it difficult to determine whether a sound is systolic or diastolic There is usually little difficulty if, simultaneously with auscultation, a finger is placed on the internal carotid artery In this way the first sound can usually be timed with accuracy, and then the second, so that adventitious sounds can in their turn be localised Attention must be focused upon each separate sound in turn, the others being for the moment excluded from notice The ability to do this comes with patient practice

The examination of the lungs and of the abdomen follows the usual routine of medical examination

CHAPTER II

SPECIAL METHODS OF INVESTIGATION

Electrocardiographic Examination

The subject of Electrocardiography is one which is complicated and in which further advances are continually being made. For this reason the interpretation of electrocardiograms must be most carefully correlated with the clinical and X ray findings, and for this reason also the interpretation of electrocardiograms is likely to be misleading in the hands of one who is not constantly evaluating them. This does not constitute a sufficient reason for ignoring the subject but *it is wise to attempt to learn to read tracings according* to the best of one's abilities. If however there is any doubt about the interpretation it is far wiser to admit ignorance than to read into the pictures interpretations which they will not subsequently bear. A full description of the electrocardiographic changes in disease does not come into the compass of the present book and for detailed information the reader is strongly advised to obtain an electrocardiographic textbook such as that of Graybiel and White (W. B. Saunders & Co. 1946).

The best introduction to the electrocardiogram is obtained by reading some clear and simple description of the heart rhythm such as Lewis's *Clinical Disorders of the Heart beat.* *

The electrocardiogram superficially may appear to be a rather complicated curve but in actual fact it represents the action currents first of the auricular and then of the ventricular muscular contraction.

The normal impulse arising in the sino auricular node travels down through both auricles and reaches the auriculo ventricular node which is normally the only passage for this between auricles and ventricles. From the auriculo ventricular node the impulse *traverses the bundle of His first the main stem then the two branches* right and left and finally by way of the terminal branches and Purkinje cells it reaches the main substance of the ventricular muscle. If the heart were a piece of simple skeletal muscle these action currents would be diphasic curves like those obtained by stimulating the frog's sartorius. But the anatomical structure of the heart

* Shaw & Sons 1933 7th edition

muscle especially that of the ventricles is somewhat involved so that the electrical curve which is the resultant of these complicated muscular activities becomes correspondingly complicated

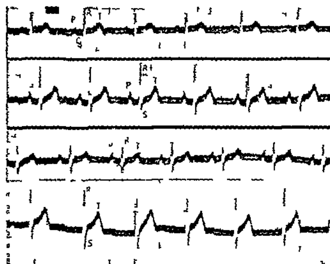


FIG 1 A NORMAL TRACING * YOUNG AD T

The P wave is upright in all three leads it is rather more prominent in Leads 2 and 3 The P R interval measures less than 0 2 second The Q wave is not visible in Leads 3 and 4 The R wave tall r n

deviation is the physiological tendency the R wave is frequently tall st in Lead 1 and the S wave correspondingly deepest in Lead 3 The P wave in Lead 4 happens to be isoelectric i e the heart happens to be so placed in reference to the electrodes that the negat i s equal as between apex and right arm so that no current passes although the auricles are contracting normally as seen by the normal P waves in Leads 1 2 and 3

It is wise to cultivate the habit of reading the electrocardiogram wave by wave The auricular curve or P wave is relatively simple and is nearly always upright But if the auricular muscle is stimulated by an impulse which arises at the bottom of the auricles near the

ventricles the direction of flow of the impulse through the auricles is *backwards or reversed* in such a case it is obvious that the P wave will be inverted An example of this is seen in some types of auricular premature beat If the left auricular muscle is hypertrophied as in mitral stenosis the P wave is often enlarged and may be notched If the normal auricular impulse is superseded by an abnormal auricular rhythm as in auricular fibrillation in which condition there is a stimulation of the auricular muscle from a focus which varies in its position and which emits stimuli at about four hundred per minute the original normal impulse is continuously overwhelmed and is thus abolished Thus in auricular fibrillation the regular normal P wave is never seen

The next point to examine is the P R interval This measures the time interval between the start of auricular systole and the start of ventricular contraction It extends from the first rise of the P wave to the first indication of the QRS complex The P–R interval normally varies between 0 12 and 0 2 of a second The interval may be increased as in heart block or decreased as in nodal rhythm (Fig 2)

The QRS T waves represent ventricular systole This part of the curve has a complicated shape but must be considered as a whole It must be remembered that the ventricular electrocardiogram represents a resultant of electrical forces The ventricles left and right contract simultaneously but in opposite directions Thus the sum of their action currents is considerably smaller than if an impulse started in the left ventricle and travelled straight through the ventricular muscle to the further border of the right ventricle or vice versa An example of this latter state of affairs is seen in premature beats where an ectopic focus does in fact originate an impulse at some place outside the proper channel of stimulation so that the contraction traverses the ventricles from left to right or from right to left This is why the normal QRS T is of a certain definite size for any given patient but if ventricular premature beats occur the electrical curve of these is often in some leads two or three times greater than the normal ventricular complex The QRS part of the ventricular curve should measure less than 0 1 second in duration *An increase above this indicates some deficiency in the conducting* powers of the bundle of His so that transmission of the impulse is not simultaneous in both branches of this structure The impulse in such cases begins to activate one ventricle in advance of the other so that there is a spreading out of electrical activity which shows itself in a wider or notched QRS This is known as a bundle branch defect

Normally the relative size of the R and S waves gives an indication as to the distribution of muscle-mass The R wave should be tallest in Lead 2, and the S wave should not greatly predominate in any lead *When the R wave is tallest in Lead 1 and the S wave simultaneously is deepest in Lead 3, left axis deviation is shown* The

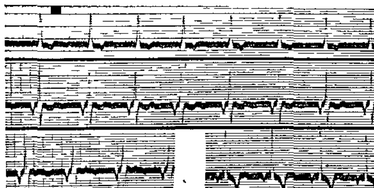


FIG 2—NODAL RHYTHM (with short P–R interval)

This is an abnormality of cardiac mechanism which has no especial clinical significance It may be found in individuals whose hearts are otherwise normal in all respects The condition is produced by the presence of a pace maker near to the position of the auriculo ventricular node Such a pace maker in some cases produces regular stimuli at a high rate causing an attack of nodal paroxysmal tachycardia In other cases of which the tracing is an example the rate is normal or slow Nodal rhythm with bradycard a may occasionally last for weeks or months at a time but is rarely permanent There is usually no associated heart d sease Nodal paroxysmal tachycardia is in all clinical respects identical with other forms of auricular paroxysmal tachycardia The short P–R interval is due to the proximity of the new focus to the auriculo ventricular node The new impulses reach the auricle and ventricle nearly simultaneously In some such cases they may actually reach the ventricle first

reverse of this, when the S wave is deepest in Lead 1 and the R wave is tallest in Lead 3 indicates right axis deviation

The next point to stress is the level of that part of the tracing immediately following the S wave In the normal electrocardiogram after the QRS waves the curve momentarily returns to the normal resting diastolic level before the T wave begins In certain conditions especially in coronary occlusion during the acute stage this so-called S–T part of the curve remains elevated or depressed and merges into the elevated or depressed T wave, without returning to the diastolic or resting level If the S–T is elevated in Lead 1 it is usually depressed in Lead 3 and vice versa, an elevated S–T in Lead 1 with depression in Lead 3 is seen in acute anterior coronary

infarction, conversely, depression of S–T in Lead 1 and elevation in Lead 3 indicates posterior infarction This is usually known as Pardee's sign

The next point to look at is the direction of the T waves The normal T waves are upright normally in Leads 1 and 2, and commonly upright in Lead 3 If the T waves are depressed or inverted in Leads 1 and 2, this constitutes a definite abnormality

Electrocardiograms were for many years taken through the three standard limb leads only Lead 1 is obtained from the right arm and left arm, Lead 2 from the right arm and left leg, and Lead 3 from the left arm and left leg Each of these leads gives a picture of both auricular and ventricular electrical activity, but each lead gives this picture from a different aspect One lead does not pick out auricles or another the left or right ventricles, as is still erroneously stated in some textbooks It is as though an individual were attempting to define the shape of a small island in the middle of a pond, and for this purpose examined the somewhat remote island from three fixed points of view The accumulated experience of many years has enabled accurate conclusions to be arrived at as to what should constitute a normal Lead 1, Lead 2, and Lead 3

A more recent step was the introduction of Lead 4R which is a lead taken from the right arm (or left leg) to the apex Since, however, this lead gave an inversion of the T wave as a normal finding, the British Cardiac Society, in agreement with the American Heart Association, agreed to reverse the electrodes in these leads, so that the normal T wave in Lead 4 should be upright, as is the case in Leads 1 2, and 3 This is arbitrary but useful Briefly, Lead 4R gives information by amplifying considerably those similar changes of a small degree and direction which may be visible in Lead 1 Further chest leads have been subsequently developed, and are valuable in the hands of experts

These additional precordial leads are analogous to the routine Lead 4 but the chest electrode instead of being applied to the apex, is applied in the following series of positions (1) at the border of the sternum in the fourth right intercostal space, (2) at the border of the sternum in the fourth left intercostal space, (3) half-way between the sternum and the mid clavicular line, where this line bisects the line drawn from (2) to the apex beat, (4) the mid-clavicular line where this bisects the line drawn through (2) and (3) (CR 4), (5) half-way between the mid-clavicular line and the anterior axillary line at a point horizontal with (4), (6) in the anterior axillary line at this same level The usual method of description of these

leads is by the use of the letter "C" representing chest lead and "R" or "F" representing the right arm or the left foot as being the situation of the indifferent electrode C R 1 therefore would represent the precordial lead where the electrodes were placed to the right border of the sternum and on the right arm respectively The interpretation of the special chest leads is as yet a matter for expert opinion

Although many electrocardiograms need expert interpretation

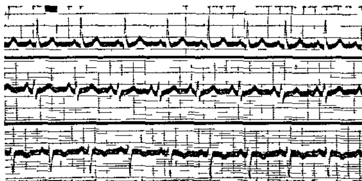


FIG 3—A NORMAL TRACING FROM A MIDDLE AGED WOMAN WITH A TRANSVERSELY PLACED HEART

The R wave is tallest in Lead 1 and the S wave deepest in Lead 3 This signifies left axis deviation which is physiological in middle aged and elderly people Inversion of the T wave in Lead 3 with left axis deviation in the absence of other abnormalities indicates a horizontally placed heart and a normal electrocardiogram The P wave may be similarly inverted in Lead 3 in such cases

there are certain abnormalities which can be easily read from the print

Left axis deviation (Fig 3) shows an R wave taller in Lead 1 than in the other leads and an S wave deeper in Lead 3 than in the other leads It is physiological in elderly people with horizontally placed hearts Right axis deviation shows an S wave deepest in Lead 1 and an R wave tallest in Lead 3 It is physiological in young children

The P–R interval is normally less than 0 2 of a second and the QRS interval less than 0 1 of a second In heart block the P–R interval can be easily measured and any increase in it can be determined If the heart block has caused a dropping out of ventricular beats the regular P waves will be seen to occur without the following QRS complex

Bundle branch block shows prolongation of the QRS wave, which

enables the rays to be cut down to an exceedingly narrow vertical strip which can be moved so that it just impinges on one or other border of the heart These rays pass forward at right angles to the screen from behind the chest and they can be adjusted to graze the heart either in systole or diastole as is required By this means the left and right borders of the heart can be outlined by a grease pencil upon the front of the screen and if necessary the whole outline of the organ can be accurately reproduced Similarly the size and shape of the thoracic cage usually in a state of ordinary inspiration can be defined An exact measurement can thus be taken and the transverse diameters of both heart and chest either antero posteriorly or in the right or left oblique positions Measurements so taken are accurate to within 0 5 cm and will be found to agree with great constancy if the heart is measured periodically provided the organ does not change in size itself

It must be remembered that the shape of the thorax varies greatly from individual to individual and this will itself cause some variation in the size and shape of the heart If the thorax is unusually deep from back to front the heart will appear to be large in relation to the transverse diameter whereas in fact it is not large considering the total thoracic volume Conversely if the thorax is abnormally shallow the heart may be compressed as it were or displaced to the left so that an impression of enlargement may be given where no true enlargement exists

Further facts must be taken into account in assessing the size of the heart Some tall and asthenic individuals have unusually narrow vertical hearts and stocky rather obese types are apt to have horizontally placed hearts which at first give an impression of enlargement The narrow vertical heart may enlarge and still remain within the average normal size measurement Another difficulty in obese patients is the presence of small triangles of epicardial fat both to the left and to the right at the cardiophrenic angles

It is thus fairly obvious that although gross abnormalities of the heart are easily diagnosable after a short experience of X ray screening yet there are many pitfalls which make border line cases difficult to assess

Information which can be obtained from X ray screening enables the size of the heart as a whole to be judged and also makes it possible to determine variations from the normal which may occur in any chamber of the heart or in the great vessels Serial measurements made in the same patient at intervals of time are of the greatest value

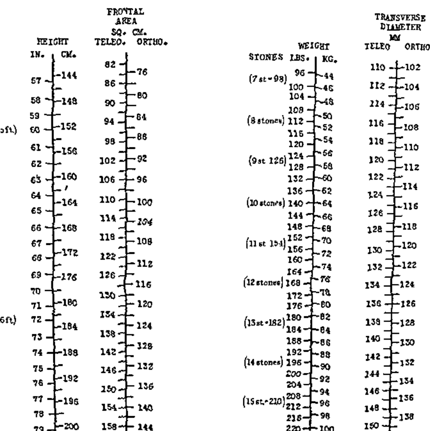


FIG 6 —TELEO FIGURED FOR QUIET RESPIRATION
Teleo area – ortho × 111 per cent Teleo TD – ortho × 108 per cent

Add 10 per cent of the patient s age to the transverse diameter of the

figures on the right transverse diameter' column refer to the orthodiagraphic measurement and those on the left to the measurement seen on a film in the same patient with quiet respiration

The nomogram is reproduced by the courtesy of DR CHESTER KURTZ from White s *Heart Disease*, with the author s permission

In order to allow for the influence of body-height and body-weight upon cardiac size valuable tables have been devised One of these in the form of an easily read nomogram, is that of F J Hodges

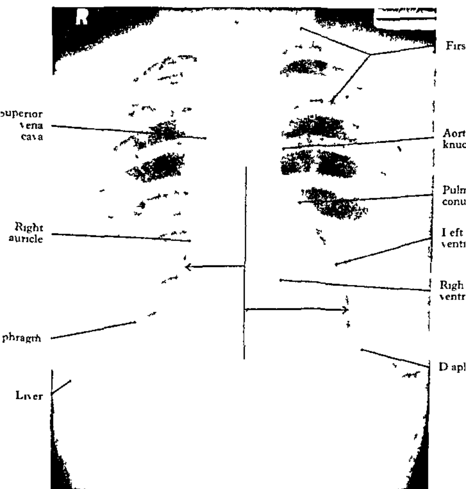


FIG 7 — NORMAL HEART YOUNG ADULT FEMALE ANTERO POSTERIOR VIEW
Transverse cardiac diameter — 11 7 cm

and Eyster and published by White (Fig 6) Although the heart size as a whole may be determined by transverse measurement, and *compared with the transverse diameter of the thorax*, greater accuracy will be obtained if this is related to the height and weight of the patient However it can be stated as a generalisation

that the transverse diameter of the heart should be less than half the transverse diameter of the thorax

A further complication underlying the necessity for regarding cardiac radiology as one factor only in the whole examination of the case is that the presence or absence of organic disease, as judged by other methods of examination, does not necessarily run parallel with the presence or absence of X-ray evidence of disease For instance *early mitral stenosis may be easily diagnosed clinically before radio* logical changes take place The same may be true of early lesions of the aortic valve Conversely a heart which appears to be normal radiologically, both as regards size and shape, may already be seriously diseased by coronary atheroma One such patient had so *severe and persistent an angina of effort that surgical alleviation* was planned The heart was normal radiologically and electrocardio graphically (Leads 1, 2, and 3) but he died suddenly a few days before the date of his sympathectomy, and at the post mortem coronary atheroma was present of so advanced a degree as nearly to *block the lumen of each coronary vessel*

In certain conditions where the presence or absence or the extent of pulsation is to be determined, kymography is a valuable X-ray diagnostic method This requires special apparatus, and specialised skill in reading the film By its use, for example, the *absence of pulsation in a part of the left ventricle, which has* resulted from previous infarction, can be demonstrated It is also possible to measure and to time the pulsation of a shadow thought to be possibly aneurysmal, possibly auricular, possibly ventricular

The antero posterior view of the heart film (Fig 7) shows *normally, on its left border, the rounded shadow of the aortic* knuckle at its highest part, below this the slightly convex curve of the pulmonary conus, then sweeping out from this to the left and down to the diaphragm comes the border of the left ventricle On the patient s right side from top to bottom of the shadow the following structures are seen at the top the rather shadowy straight outer edge of the superior vena cava, below this the slight curve of the ascending aorta, and from this to the diaphragm the very prominent curve of the right auricle

The right anterior oblique view (Fig 8) shows to the patient's left, from top to bottom the rather small curve of the ascending aorta, and from this to the diaphragm the right ventricle A clear space should intervene between the right ventricle and the shadow of the thoracic cage On the right side of the heart, or on the left edge of the heart shadow as seen by the observer, the border is nearly

straight only slightly curved in a general uniform way from top to bottom Between this curve and the vertebral column lies the posterior mediastinum which presents a clear space whose antero

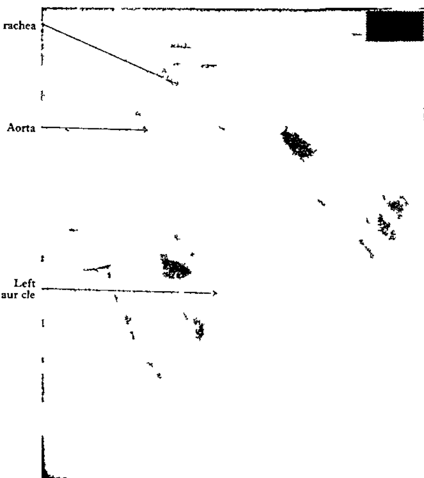


FIG 8 NORMAL HEART ELDERLY INDIVIDUAL RIGHT ANTERIOR OBLIQUE VIEW
Note dens ty of aort c shadow

posterior borders run almost parallel The posterior border of the heart in this position is composed of left auricle above and right auricle below auricular enlargement is seen as a bulge which extends posteriorly into the otherwise clear mediastinal space The swallowing of barium cream will outline the œsophagus which runs

close behind the heart The œsophageal shadow can in this way be used to accentuate the visibility of auricular enlargement

The left anterior oblique view (Fig 9) in which the patient s

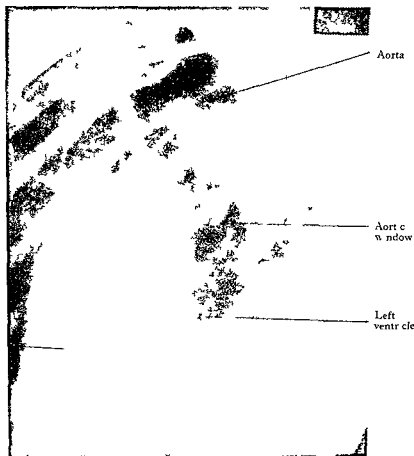


Fig 9—Nor ial Heart Sa e Pat ent as Fig 8 Left Anter or Obl que Vie

Arrow on left po nts to the lef breast shadow the r ght ventr cle s just med al to th s

left chest is applied firmly to the screen shows the following structures on the anterior border of the heart shadow At the top the curve of the ascending aorta which runs clear of the trachea leaving a small light area known as the aortic window Below the as cending aorta and anteriorly a curve sweeps down to the diaphragm

which at the top is caused by the right auricle and below by the right ventricle On the posterior part of the heart shadow in this position, correspondingly lie the left auricle above and the left ventricle below The left ventricular shadow should be clear of the vertebral column

Pathological changes in the radiological outline of the heart are on the whole matters for expert decision, but certain abnormalities which are relatively simple can be easily recognised

Pericardial disease may show itself either as pericardial effusion or as constrictive pericarditis If there is an effusion the outline of the cardiac shadow becomes more globular as a whole, and the curve of the outline due to the effusion is most prominent at the bottom of the heart shadow on both sides As the effusion increases these lower curving outlines extend higher, and obscure progressively the normal irregularities of the cardiac silhouette Calcification of the pericardium is seen most easily in the oblique or lateral positions, and upon the anterior or the under surfaces of the ventricles

Left ventricular enlargement is seen by an extension of the lower part of the cardiac shadow on the left side in the antero-posterior position Hypertrophy of the left ventricle causes this area to be more spherical, and dilatation causes it to extend further to the left and further downwards Left ventricular enlargement is also confirmed by observing that in the left oblique position the posterior part of the heart shadow overlaps the vertebral column

Enlargement of the right ventricle is also seen in the left oblique position, and in this case the right border of the heart extends further forwards towards the chest wall than is normal In the right oblique position enlargement of the right ventricle is also seen to the front of the heart silhouette, and extends similarly forwards towards the chest wall Enlargement of the right border of the heart in the antero posterior position occurs as a result of almost every form of enlargement of the heart, or part of the heart, and this is usually the result of displacement of the right auricle further to the right

Enlargement of the left auricle is best seen in the right oblique position by the help of a barium swallow Mitral stenosis is the commonest cause of gross enlargement of the pulmonary conus, which is visible above the shadow of the left ventricle on the left border of the heart in the antero-posterior position Usually there is also some enlargement of the right ventricle and sometimes of the right auricle, so that in this condition the right border is further to the right than normal

A very prominent pulmonary conus is also frequently visible in patent ductus arteriosus

The shadow of the aorta is frequently more prominent than normal, and this is usually the result of elongation due to aortic atheroma The ascending aorta seen just above the shadow of the right auricle becomes more prominent, and so does the aortic knuckle which in such cases extends further towards the direction of the patient's left shoulder Sometimes instead of the aorta sweeping inwards from this enlarged aortic knuckle towards the mid-line it extends down from this position in a vertical direction These views of aortic enlargement are not infrequently mistaken by unskilled observers for aortic aneurysms It is wise to read as much as one can from screening or films, but in the absence of considerable experience it is still wiser to be dogmatic only upon things well within one's extent of knowledge, and to avoid trying to form decisions in excess of real experience

Section II

HEART FAILURE

CHAPTER III

THE NATURE OF HEART FAILURE

THE function of the heart is to pump around the body the fluid which contains the oxygen and food necessary to the life of the tissues It also transfers various waste products from these tissues to specialised organs which remove or neutralise them

If the pumping action of the heart fails the rate of circulation of the blood is correspondingly retarded Oxygen and food arrive more slowly, and waste products depart more slowly. The organs react to these changes unequally, according to the urgency of their dependence upon the oxygen and food supply, and according to their sensitivity to the presence of waste substances which should be removed Lack of oxygen is a most important consequence of heart failure, the brain reacts within a few seconds, other organs more slowly

A second principle of great importance is that the heart cannot fail as a pump without causing a change in the anatomical disposition of the fluid it is designed to propel Diminution in the volume of the circulating blood will occur in front of the failing chamber of the pump, and accumulation of blood will be found behind it

Contrary to popular conception heart failure is rarely sudden In most cases the pump's action gradually becomes less efficient Warning of this deterioration is usually prolonged Even when serious disease strikes at it suddenly, the heart usually survives the immediate blow and struggles on in its damaged state, often for many years

These various factors, singly or in combination, will explain the symptoms and the signs of most examples of cardiac failure

The heart is a complex piece of mechanism, and is a machine which is pumping blood through two separate systems, the systemic circulation and the pulmonary circulation These function in parallel, but they differ in their blood pressure and in their vascular area The blood pressure in the systemic circulation is four or five times greater than that in the pulmonary system, and it follows that the force and muscle-mass of the two ventricles vary correspondingly

Disease may disturb this balance either by reducing the force of one ventricle as compared with the other, or by introducing a mechanical insufficiency, so as to increase the circulatory defect of one side of the heart out of proportion to the strain upon the other Thus the heart may fail as a whole, from some cause which produces an even distribution of weakness throughout the organ, or failure may attack chiefly the left side or chiefly the right

Left-sided failure is usually the result of mitral stenosis which *interferes with the supply of blood to the left ventricle, or of coronary* disease which interferes with the nutrition of the left ventricle In left-sided failure the balance of the circulation is upset, so that accumulation of blood is produced in the lungs out of proportion to the general balance

Failure predominantly of the right ventricle is rare, but when it occurs its physical signs are striking, depending as they do upon a marked absence of any pulmonary congestion It is, of course true that cases of chronic left-sided failure ultimately involve a secondary failure of the right side in addition, but in this case the classical picture of pure right sided failure is absent Briefly, pulmonary congestion predominates in the picture of left sided failure, and absence of pulmonary congestion is the mark of pure right sided failure

In addition to congestion, the nutrition of the tissues and of the capillary endothelium may suffer in all types of severe heart failure The result of this is that there is an accumulation of blood in the capillary bed, and also an increased intracapillary pressure causing an exudation of fluid from the circulatory system into the tissues This is known as œdema

Pulmonary œdema shows itself by the presence of fluid in the pulmonary passages, either manifested as râles usually basal and crepitations, or by the coughing up of œdema fluid straw-coloured or blood stained

Systemic œdema shows itself by an obvious swelling of the subcutaneous tissues, the position of such swelling being largely determined by gravity In addition to these obvious changes, *congestion and œdema occur in the liver, kidneys and other internal* organs Heart failure with manifest systemic œdema is known as *congestive heart failure*

Cause of Heart Failure

It may be stated that a healthy heart never fails It is a common lay conception that the condition in which an athlete is 'rowed out'

or 'run out' is brought on by insufficient cardiac power It is in reality due to vasomotor or psychological causes

Heart failure may be produced by the following agencies·

(1) Factors which are blood-borne, and which circulate evenly, and affect all chambers of the heart in a more or less equal manner Examples of these are anæmia, bacterial toxins, or nutritional and metabolic defects

(2) Abnormalities of rhythm, like paroxysmal tachycardia or auricular fibrillation, may interfere with the proper output from the heart These also in most cases have a general effect upon both sides of the heart equally

(3) Pericardial disease may be a handicap to proper heart function Where such disease takes the form of pericardial effusion or of chronic fibrous thickening and adhesions the chief effect of it is to interfere with the proper filling of the auricles and the ventricles, thus reducing their output

(4) Valvular defects may interfere with the proper action of the heart as a pump, by obstructing its action as in mitral stenosis, or by adding to its work as in aortic regurgitation Since the majority of such lesions affect the left auricle and ventricle more than the right, it is usual for left sided heart failure to predominate in these cases

(5) Vascular disease has a variable effect on the cardiac efficiency It is clear that an increased blood pressure imposes additional work A normal ventricle can cope with this but to a weakened muscle the added pressure is a handicap

These are the chief general factors concerned in the production of heart failure and it is useful to reconsider them in more detail

Anæmia may influence the state of the heart muscle very greatly The best example is the heart of pernicious anæmia Post-mortem evidence shows that in this condition there is produced the most obvious and the most widespread fatty degeneration of heart muscle But anæmia of any kind may have a similar action The effects of anæmia are produced upon the heart as a whole, so that in such cases there is an equal failure of both ventricles, and an absence of pulmonary congestion Thus dyspnœa and orthopnœa, the symptoms chiefly dependent upon pulmonary congestion, are not very prominent

Another cause of myocardial failure is widespread *pulmonary disease*, whether acute or chronic Heart failure is the chief danger in pneumonia, and the anoxæmia present is probably as important as the bacterial poison Chronic failure from anoxæmia may occur in

pulmonary emphysema Here the loss of full pulmonary efficiency reacts unfavourably on the health of the heart muscle

Anæmia may be produced locally in the substance of the heart muscle by interference with the flow of blood through part of the *coronary circulation* Atheroma is a common cause of this, but it may occur in syphilis If the diminution in oxygen supply is sudden, as in coronary thrombosis, the muscle may die and rupture, and even a less degree of obstruction may be sufficient to cause fatal heart failure In other cases the interference with blood flow is much more chronic, and produces a slow degeneration of some area of the heart muscle Since the lesion is local the myocardial change is also local, and affects the bigger muscle-mass of the left ventricle more frequently than the right, causing pulmonary congestion

Certain *bacterial poisons* have a particularly severe influence upon the state of the heart muscle The toxin of the Klebs Loffer bacillus produces an acute diphtheritic myocarditis in five or six days from the onset of the disease in a severe case The effect is seen in wide spread fatty degeneration, equally distributed throughout both sides of the heart The heart muscle is then extremely flabby, and shows signs of disease both clinically and on post mortem examination Sudden death may occur, although there was little shortness of breath or orthopnœa during life owing to the absence of pulmonary congestion Other diseases which cause severe acute myocardial change are acute rheumatism, pneumonia, and typhoid fever Rheumatism may attack the heart repeatedly during a period of many years leaving behind it a slowly increasing extent of injured heart muscle In such cases this gradual muscular degeneration following inflammation is a more serious matter than is the associated valvular lesion

The commonest *metabolic abnormality* which affects the state of the heart muscle is seen in disease of the thyroid gland Thyroid deficiency not only causes enlargement of the heart, which subsides after treatment, but also produces signs and symptoms of heart failure, though these are usually slight in degree Hyperthyroidism also is very commonly associated with signs and symptoms of heart failure, both to the congestive and pre-congestive type It is sometimes suggested that the persistent tachycardia is sufficient cause for these, but it is possible that thyroxin not only increases the metabolic rate and the normal activity of the heart, but in addition has an irritative effect upon the myocardium Beriberi and lesser degrees of vitamin B_1 deficiency may also cause general failure of the heart muscle as a whole

Various *cardiac irregularities* affect the function of the heart

The most obvious example is seen in a prolonged attack of paroxysmal tachycardia In such a case the heart is suddenly driven at a much increased rate for example 140 beats per minute, for a length of time which may extend to four or five days Here evidence of heart failure rapidly appears, and finally the picture may be one of severe congestive failure with much œdema, enlargement of the liver, ascites, and albuminuria The direct cause of this failure is the greatly increased heart rate which abruptly reduces the total period of time available for diastole, so that cardiac filling is seriously impaired, and cardiac output correspondingly diminished for minutes, hours, or days Similar examples are found in paroxysmal auricular fibrillation and to a less extent, but for a more prolonged period, in auricular flutter

Ectopic rhythms may influence the heart function in other ways Instead of the onset of auricular flutter or fibrillation or tachycardia, ventricular fibrillation may result from the presence of severe ventricular disease This causes sudden cessation of the flow of blood, and sudden death

Heart block may be associated with a different form of sudden heart failure In complete heart block the heart contracts slowly at a rate of 30 to 40 beats per minute, the rhythm being produced by a pace-maker in the upper part in the bundle of His Should this pace-maker itself be influenced by disease or drugs it may cease working, temporarily or permanently In the former case a syncopal attack may occur, in the latter case the patient dies suddenly

The efficiency of the heart may also be reduced by structural changes in it or around it, which through *mechanical factors* interfere with its intake of blood or its output, or which may handicap the onward flow from chamber to chamber, and which in other cases may allow some backward leak to occur These defects affect the cardiac function and consequently induce variations in cardiac structure, which are sometimes compensatory

Mechanical handicaps to the heart's action may be pericardial, endocardial, or vascular

Pericardial effusion, whether acute or chronic, can cause a progressive increase in the pressure inside the pericardial sac When this pressure begins to approximate to that present within the auricles and great veins there is a rapid interference with cardiac filling [illegible]

[1] obviously distended

Chronic pericarditis, in which the pericardial layers are thickened and frequently adherent, has a much slower but an equally severe effect on the filling of the heart The ventricles are unable to distend so as to accommodate the normal quantity of blood, and the ventricular output is reduced This produces heart failure of the usual type, progressing to œdema and the other signs of congestive failure Here also the veins in the neck are often visibly distended

Both these varieties of pericardial disease may affect the right ventricle as much as the left, and may therefore cause venous distension and systemic œdema out of proportion to shortness of breath and orthopnœa

The great majority of cases of acquired *valvular disease* involve the left side of the heart Their effect upon the circulation depends upon the extent to which the efficiency of the left ventricle is affected This chamber is able to compensate by hypertrophy for both types of disease of the aortic valve, and for mitral regurgitation It fails to meet the situation only in mitral stenosis, because the narrowed valve deprives it of an adequate quantity of blood to expel The left auricle is a thin chamber incapable of great muscular effort and it compensates poorly for mitral stenosis But a small degree of mitral stenosis is compatible with a normal circulation rate

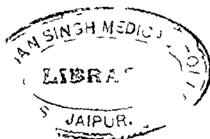
A successful jockey was found to have severe incompetence of the aortic valve He had been examined twenty years previously by Sir James MacKenzie who told him that he had an aortic regurgitation During that period he had risen to the top of his profession in spite of the valvular defect A recruit from St Albans Medical Board living in Hertfordshire was found to have classical signs of aortic stenosis His week end hobby was to visit his relations in Birmingham on a push-bicycle from time to time A Durham miner who had no symptoms, and a girl who had done well in competitive sports as a sprinter, were both found to have definite mitral stenosis, though in these cases it is clear that the narrowing must have been slight

Thus in valvular disease, as in nearly all other varieties of heart disease, the problem is to estimate the health of the heart muscle, and its capacity for adjusting itself to the new physical conditions imposed upon it by the defect

It would appear to be a reasonable assumption that a persistent and considerable *increase in the blood pressure* would be likely to impose a burden upon the heart muscle sufficient to interfere with its proper function This is not the case unless the heart muscle is itself unhealthy A healthy heart muscle, well supplied with oxygen

and food, is capable of standing up to almost any burden that can be placed upon it When the systemic blood pressure is raised, the increase occurs sufficiently slowly to enable the left ventricle to compensate, and since the pressure during diastole is above the normal in such cases, the coronary flow, which occurs in diastole, is actually improved and increased by this raised pressure

If the pulmonary blood pressure is increased, there is no such compensatory effect upon the coronary flow, and this may be a reason why right ventricular failure is not uncommon as a complication of chronic bronchitis and emphysema



CHAPTER IV

THE SYMPTOMS OF HEART FAILURE

Shortness of breath *is the symptom of paramount importance in disease of the heart* If shortness of breath is not present when the patient is undergoing normal exertion it may be stated that there is no heart failure The amount of exercise or exertion which produces dyspnœa and other cardiac symptoms may be estimated in different ways Many attempts have been made to standardise exercise tolerance tests In these tests a fixed amount of exercise, measured in foot-pounds, is performed by the patient in a specific time The effect upon the heart rate is noted, by measuring this before and immediately after exercise, and also by noting the length of time taken for the accelerated heart to return to its original rate During such tests the physical reaction of the patient in other respects is also noticed

More information can probably be obtained by taking a really careful history Patients should be asked the extent of their usual amount of exercise How much do they walk, on the flat, or uphill, and with what results as regards shortness of breath? Similar enquiries are put with regard to games, stairs, and running A woman is asked whether childbirth had any effect upon her subsequent ability to perform her usual activities In this way it is possible to judge fairly accurately the amount of exercise a patient can undertake

A second point to ascertain clearly is the rapidity of onset, over a period of days, weeks, months, or years, of any shortnest of breath the patient may complain of How long ago was it thas these activities could be performed without symptoms, and what is the present extent of such symptoms? In this way the speed of development of failure can be assessed

There are a number of theories as to the physiological cause of dyspnœa, but the most plausible is that the symptom is proportional to the extent of pulmonary congestion Transient acidosis caused by an increase in the amount of carbon dioxide in the blood, or of lactic acid liberated from the muscles and present in the blood, has also been suggested as a cause But, although the influence of these factors may exist, it is probably slight Anoxæmia has also been

proposed as an explanation for dyspnœa, but this theory is now discredited

Dyspnœa may occur in some patients when they are at rest, as a result of myocardial disease This is of the usual type when failure is acute and severe, but there are two additional varieties of dyspnœa at rest which require description When the left ventricle is severely damaged it would appear that sudden pulmonary congestion periodically occurs in some patients, especially at night This congestion produces a reflex spasm of the bronchioles, and sets up an attack which is indistinguishable from that of spasmodic bronchial asthma It is called *paroxysmal dyspnœa* or *cardiac asthma* The patient s chest becomes tight and wheezy He can no longer lie flat, and may have to sit forward grasping the back of a chair or some other fixed object This enables the accessory muscles of respiration to be fixed and brought into play to help the embarrassed breathing The attack does not usually last as long as that of true spasmodic asthma for it rarely exceeds one or two hours at the outside, and is most often an affair of minutes Nevertheless it is extremely distressing and is usually a sign of serious myocardial disease

Another variety of dyspnœa at rest is *Cheyne-Stokes's respiration* In most cases this periodic increase and decrease of the depth of respiration passes unnoticed by the patient, but in a few cases the violence of the hyperpnœa is sufficient to distress, to disturb sleep, or to frighten Cheyne-Stokes's respiration, however, is probably a manifestation of vascular disease affecting the respiratory centre, and is not evidence of cardiac trouble This is called *periodic dyspnœa*

Orthopnœa, like dyspnœa, generally indicates pulmonary congestion and therefore left-sided heart failure In all probability it is due chiefly to over-distension of the lungs with blood, but congestion and enlargement of the liver may also be a factor For it is clear that the force of gravity in a more upright position, is likely to impede to some extent the return of blood from the vena cava, and will also allow the enlarged liver to fall away from the thorax The pulmonary congestion probably causes both dyspnœa and orthopnœa by means of a pulmonary reflex

Thoracic aneurysm and new growth may also cause orthopnœa

Sighing

This symptom is frequently considered by lay individuals to be evidence of heart disease It is sometimes described by patients as "shortness of breath " When questioned they will state that there

does not seem to be sufficient room in the chest for air, or that they find it hard to get in a full breath. Sighing is not in any way evidence of heart disease, but is found in patients with hypersensitive nervous systems, with anxiety troubles, or in definite psychotics. Its presence is almost evidence of the absence of organic heart disease.

Irregularity of respiration, in depth and rhythm, is well shown in these patients on respiratory graphs and tracings.

Tachypnœa or Rapid Breathing

Certain individuals of a neurotic temperament have attacks in which there is a "functional" increase in the speed and depth of respiration. The hyperpnœa often continues sufficiently long for a state of alkalosis to be produced. This shows itself eventually in the hands and feet by tingling and muscular contracture, and even by tetany. Before this stage is reached the heart rate is often greatly increased, and the pulse chart may show peaks which suggest paroxysmal tachycardia. The increased rate frequently reaches figures of about 150, and the tachycardia lasts for a considerable length of time. Sometimes this increased heart rate together with apparent shortness of breath are the predominant features, so that some form of organic heart disease may be erroneously suspected.

Cough

Cough is occasionally a cardiac symptom, especially if there is a state of chronic pulmonary congestion. Some patients with mitral stenosis, for example, will complain of cough which is brought on by exertion. But cough is a frequent symptom in asthma, and asthmatics not infrequently get an increased wheezing in the chest on exertion, especially in cold or windy weather. It is important in such cases to disentangle the three symptoms: cough, wheezing, and breathlessness. The true asthmatic will complain chiefly of the wheezing and tightness which is the cause of both the dyspnœa and the cough, and usually he has no enlargement of the heart.

In chronic bronchitis myocardial degeneration may ultimately occur. In such cases the history will make clear the fact that "winter cough" preceded by many years the onset of dyspnœa. Here the right ventricle is most affected.

Pulmonary tuberculosis also in some cases is very chronic, and the fibrosis produced may be extensive. Cough and increasing shortness of breath is then complained of out of proportion to the signs and symptoms of toxæmia due to the infection, and a cardiac condition may be erroneously suspected.

Paroxysmal dyspnœa is another cause of cough in cardiac subjects The sudden attack, usually nocturnal, may be terminated by the coughing up of considerable amounts of frothy pink or yellow albuminous sputum This is due to sudden acute pulmonary œdema

Pain

Pain is not a symptom of heart failure, but since it is found in patients with cardiac symptoms it needs to be considered Coronary pain is usually central, across the upper chest, radiating to the arms, and exactly proportional to exercise It may occur without much evidence of actual myocardial failure Functional pain, sometimes thought to be cardiac, is of two main varieties It is usually a dull heavy ache or feeling of fullness over the left chest It follows exercise rather than accompanies it, and frequently persists for some hours at a time Another functional type of pain is the sharp stab in the left chest which is momentary, but which may precede the dull constant ache

Pain may be felt over the right hypochondrium in rapidly *increasing congestive failure when the liver is congested The pain is* due to the stretching of the liver capsule

Palpitation

Palpitation is a better guide to the nervous sensitivity of a patient than to the organic state of the heart It is usually absent in organic heart disease, except in paroxysmal tachycardia and at the onset of auricular fibrillation But mitral stenosis is an exception to this rule

When palpitation is complained of it is essential that the history should be very clearly taken When the palpitation is nervous in origin it is accentuated by the physiological causes of increase in the heart rate Excitement and emotion, as well as exercise, are the usual causes The heart does not abruptly increase its rate, but the palpitation runs parallel with the cause In palpitation due to paroxysmal tachycardia the onset is abrupt

Indigestion is another source of palpitation, and some people who are sensitive to tobacco, alcohol, or coffee complain of this as a cause of sleeplessness Again, premature beats occurring as isolated throbs, bumps, or stabs may be described by the patient as palpitation

Exhaustion

Nearly all patients suffering from heart failure, whether slight or severe, complain also of fatigue or exhaustion But this symptom, like palpitation, is a more characteristic manifestation of nervous

sensitivity The essential point to clarify in the history is whether shortness of breath is more prominent than exhaustion, or whether exhaustion is more complained of than dyspnœa In the former case, organic heart disease is likely to be present, whereas the reverse is found in effort syndrome or neuro circulatory asthenia, which is a disorder of somatic or psychological nervous function

Exhaustion which is accentuated by standing is common during convalescence from infections, and in individuals suffering from fatigue of any kind Here the mechanism is probably vasomotor, and due to transitory and partial cerebral anæmia In such patients a change from the recumbent to the erect position produces an increase in the heart rate and a fall in the blood pressure

Giddiness

Giddiness is also usually a vasomotor symptom, and is then accentuated by standing up after sitting or lying down There is, *however, one variety of giddiness which is occasionally taken for a* heart attack This is aural vertigo resulting from vestibular disease or disturbance It is common in individuals with arteriosclerosis and hypertension and may then arise from some interference with the blood supply to the vestibule A careful history in these cases reveals that the presumed fainting attack was not accompanied by any loss of consciousness, but that the room or surroundings began to revolve, producing a frightening sensation of physical instability, not infrequently associated with tinnitus and vomiting Deafness has been present for a long time in some of these patients Movement of the head accentuates the symptoms

Fainting

Fainting is another symptom commonly but erroneously thought to denote heart disease People with heart disease actually do not faint The cause is cerebral anæmia, usually from loss of vasomotor control The true fainting attack is in most cases not abrupt, so that there is some warning even though this may be brief Things become blurred and distant before the patient loses consciousness The patient feels cold and clammy, and sweats The blood pressure falls and the heart rate is slowed Epilepsy must always be borne in mind, and evidence as to jerking movements, incontinence of sphincters, and a family history of fits must be carefully sought

There are three cardiac causes of fainting all of them rare Aortic valvular disease may produce syncope In an aortic stenosis, for instance, exercise sometimes brings this on Paroxysmal

tachycardia where the mechanism is that of auricular flutter, in which the ventricular rate jumps to 250 or so, may cause sufficient cerebral anæmia to produce fainting Stokes Adams attacks in heart block will also cause syncope from temporary standstill of the ventricles

CHAPTER V

THE SIGNS OF HEART FAILURE

HEART failure produces no physical signs in its earlier stages It is only with the advent of the congestive stage that signs become noticeable Physical signs are least prominent when the failure involves both ventricles and both circulations equally, and are most prominent in the lungs with left heart failure, and in the systemic circulation with right heart failure

Shortness of breath may be noticed in a patient soon after he has entered the consulting-room if he is ambulatory, or after the effort of undressing or dressing-up again If shortness of breath is noticed when the patient is at rest, failure is already at an advanced stage Shortness of breath which lasts for an abnormally long time is also produced in failure of moderate degree by slight exercise, such as walking up a short flight of stairs

Orthopnœa, or inability to lie flat, is perhaps an earlier sign of left-sided failure than is obvious dyspnœa

The physical examination of the heart itself gives no exact clue to failure, for a large heart may be well compensated, and valvular disease and cardiac irregularities may also be well compensated Certain deductions as to the condition of the heart muscle may be drawn from physical examination of the heart and blood pressure If the cardiac impulse is diffuse but weak it may be inferred that early heart failure may be present, or is not far distant A similar conclusion may be drawn if the first sound is of noticeably poor quality, if the heart rate at rest is persistently raised in a cardiac subject, if pulsus alternans as measured by the sphygmomanometer is observed, or if triple rhythm is noticed at the apex in hypertension

The lungs will give evidence of left-sided failure by the presence of fine râles at both bases A small pleural effusion, in organic heart disease, is similarly suggestive

When the right ventricle also fails to a considerable degree, venous congestion becomes apparent in the veins in the neck The increased venous pressure may be measured by noting the distance up the neck veins reached by this state of permanent filling The base line above which this level is to be measured is the level of the

lower edge of the upper border of the manubrium sterni Cardiac œdema first shows itself *round the ankles, and extends progressively* to the knees, thighs, and abdominal wall and lumbar region The scrotum also becomes œdematous Congestion of the liver produces enlargement of this organ, congestion of the kidneys causes albuminuria

Œdema of the Legs

It is very important to define the facts available from the history in patients showing œdema of the legs and feet Was this preceded by dyspnœa? Is it accompanied by orthopœa? Many patients have œdema of the legs and feet from causes other than cardiac failure Such patients either have no dyspnœa, or in them the swelling has existed for a considerable time before dyspnœa was noticed Prolonged standing, especially in women with poor muscular development or with tissues which seem to be disposed to the condition, may produce œdema of a considerable degree Such œdema is accentuated by warm weather, or previous to menstruation, and is especially noticed towards the end of the day During the air-raids on London when sleeping for weeks in deck-chairs was common in people who stood during the day, œdema of the legs and feet was noticed frequently in men as well as in women This was called "shelter œdema' In peace-time conditions cases of orthostatic œdema seem to be almost confined to the female sex Phlebitis, or varicose veins, possibly associated with pregnancy, also constitute a common cause of œdema of the feet Here, however, the condition may be unilateral, and if bilateral is usually more marked on one side

If ascites accompanies œdema in a patient it is essential here, also, to define accurately the sequence of events Fluid may accumulate in the abdomen under pressure, and this in its turn, by interfering with the return of blood from the legs may later cause œdema of these Examples of this are cirrhosis of the liver, new growth of the liver, and other similar mechanical conditions

Finally it is a curious peculiarity of two cardiac conditions, that ascites may occur *before œdema of the legs and feet, or if the ascites* and the œdema are simultaneous the ascites may predominate One of these is constrictive pericarditis, the other certain cases of chronic mitral stenosis In both diseases chronic hepatic congestion may give rise to some fibrosis or cirrhosis, which may be the cause of the ascites

CHAPTER VI

THE TREATMENT OF HEART FAILURE

A PATIENT with slight failure may be ambulatory, with symptoms only, and without evidence of congestion Another patient, with severe failure, may have in addition œdema of systemic and pulmonary systems, with enlarged liver, ascites, albuminuria, and other manifestations of congestive failure The two problems are different and need separate consideration But certain aspects are common to either case

An exact diagnosis is first necessary The type of heart disease present must be determined The state of the heart muscle must be investigated with particular reference to the size of the heart, and to the presence of hypertrophy and of dilation The larger the *heart the more serious, on the whole, is the outlook If information* is available, the rate of previous increase in size is helpful in prognosis

The factors causing the muscular change must be evaluated A myocardial poison may be present, like that of active rheumatism Thyrotoxicosis or anæmia may be causing injury An irregularity, such as auricular fibrillation or auricular flutter, may be overdriving the damaged organ A valvular or a pericardial lesion may be adding a mechanical handicap Hypertension may have increased the burden

In addition to these expected complexities it must be remembered that any type of illness, from peptic ulcer to acute bronchitis, causes embarrassment to an already diseased heart These factors must not be overlooked, for their rectification will help the cardiac recovery

Finally it is necessary to obtain from the patient a true account of the normal activities and exertions of life during the period preceding the illness, for readjustment of these will be a part of the present or the future treatment

An *ambulatory patient* will require a period of rest in bed, if some active condition is present, such as subacute rheumatism, severe anæmia, or thyrotoxicosis, if the onset of dyspnœa and distress has been recent and rapid, or if a period of rest in bed seems to be the only way of emphasising to the patient the importance of modifying future activities

If no actively injurious agent is adding to its difficulties, the heart will not be harmed by some exercise But this must be for some weeks reduced well below the amount which recently caused dyspnœa and distress It may then slowly and quantitatively be increased Walking on the flat is the most valuable form of therapeutic exertion, and this may be combined with, for example, an increasing amount of easy golf on a flat course, or easy work for short spells in a garden During this period adiposity must be diminished by reducing the carbohydrate intake Breathing exercises may also be of value Treatment in other respects will vary with the type of heart disease present

When the patient is ready to return to work the future programme must be laid down in detail, and a reduction must be made in the amount of exertion previously undergone Hours of work, length and ease of journey to work, leisure for meals, and the possible delegation of worries or burdens to colleagues, must be investigated Future rules of conduct, deduced from these facts, must be explicit Nor must regulation of home activities, the use of alcohol and tobacco, the size of the evening meal, hours of sleep, and sexual intercourse, be omitted from the advice given

Congestive failure requires a similar analysis as regards the presence of causative or subsidiary factors, but here a period of rest in bed is imperative, even in a comparatively mild case A severe case will require much attention to therapeutic detail The position in bed should be that of maximal comfort, this will generally be found to be one in which the back and head are supported rather high, the thighs are slightly flexed, and the lower legs are lowered Several types of "heart bed" have been evolved, in which the amount of support to the back, the degree of lowering of the legs, and the angle of tilting of the patient as a whole can be varied and suited to the individual case

The patient, however ill, should exercise the lower legs and the feet twice daily as a routine Failure to do this encourages clot to form in the popliteal veins and to spread to the femorals, whence it becomes liberated producing pulmonary embolism This complication is one of the commonest causes of deterioration, and even of death, in patients with congestive failure

The skin must be most carefully watched, bedsores are common Regular washing, friction with spirit, and dusting with powder are essential

Diet as a clinical question is often shelved airily under the heading "light," when really ill patients are concerned The

adjective "light" has many different literary meanings It may refer to specific gravity, colour, humour, or morals It should therefore not be used in classifying diet A more precise attitude of mind is necessary in relation to calorie-content, fluidity, bulk, and the effect of various foods on the functions of the stomach and bowels

The effect of diet in a patient with congestive failure should be to relieve the heart and circulation of unnecessary work, to reduce fluid intake, and to prevent a distended stomach from pressing up against the heart In a severe case, starvation for one day or for two, except for small sips of fluid containing sugar and, if liked by the patient, a few toffees or boiled sweets, will reduce the basal metabolic rate, keep the salivary ducts flushed and allow excretion of œdema fluid to begin to overtake the fluid intake For the next few days the fluid intake can be kept at 1,000 c c or even less, and the food intake can remain low, 1,000 Calories or less As immediate recovery begins the food can be increased to 1,200 Calories or so, and the stringency of the fluid ration slightly relaxed The type of food taken matters little, provided that the patient likes it, and provided that it is as solid as possible Fluid dishes—like gruels, soups, stews, fruit salads, and milk foods—should be avoided But milk should be given instead of water to quench thirst Alcohol in non-bulky rather concentrated form often helps appetite and sleep and does no harm Whisky and brandy are preferable to beer A glass of wine with meals is allowable As little sodium chloride as possible should be taken Limitation of the sodium is more important than that of the chloride Where dietetic facilities allow of it a sodium-free, or nearly free, diet is worth trying in a stubborn case of failure Extra vitamins may be necessary, if the history suggests a previous lack of adequate food

Sleep is essential in the acute stage, and for the first few nights of acute failure morphia should not be withheld Morphine sulphate, gr $\frac{1}{6}$, gr $\frac{1}{4}$, or even gr $\frac{1}{2}$, should be given by injection, the dose depending upon the size of the patient and the urgency of his need for rest. If a patient vomits when given morphia, papaveretum (B P C) gr $\frac{1}{3}$ can be given by injection, and repeated in three hours if necessary

If a soporific is required in a chronic case potassium bromide and chloral hydrate, gr 20 of each, may be given by mouth

The barbiturates are especially liable to cause cerebral symptoms in patients with cardiovascular disease Butobarbital is probably the best of these, gr $1\frac{1}{2}$ to gr 3

Paraldehyde by mouth or per rectum is often useful, m 60 to m 120 by mouth, and twice these doses per rectum

Digitalis is of course the standard drug for all cases of heart failure, but its action is predominantly powerful when auricular fibrillation is present Its use in the condition is described elsewhere (p 96) When failure with regular rhythm is present the same recommendations hold good with few modifications In cases with recent coronary symptoms digoxin should not be given intravenously, and a massive initial dosage is best avoided, for such patients sometimes react badly to it It may induce an increased ventricular irritability associated with multiple premature beats and a corresponding decrease in ventricular output If the drug is at first given to cases of chronic failure in doses of digitalis folia gr 1, six times in the first day and four times daily afterwards, its effect can be watched and the dosage reduced or modified as judged best Should a more rapid action be required digoxin in equivalent amount can be substituted for the first few days

Theophyllin-ethylene-diamine (Cardophylin) is a valuable drug in some cases of congestive failure with raised venous pressure, especially if this is due to arterial disease It is given intravenously in a dose of 0 48 gramme in 20 c c saline Injections can be given daily for a week, but one two, or three successive daily doses are often sufficient

Œdema is not only a result of heart failure, its presence acts deleteriously, in the lungs to oxygenation, and in the tissues to local physical health Its speedy removal is therefore important

The introduction of the mercurial diuretics was one of the great advances in circulatory therapeutics These drugs presumably act by slowing down the reabsorption into the tubules of the glomerular filtrate The compounds used are organic preparations of mercury combined with urea (novasurol) or with theophylline (mersalyl, neptal) They are given usually by intramuscular or by intravenous injection of ½ to 2 c c of a 10 per cent solution Suppositories have sometimes been found to be effective The interval between injections varies between two and seven days, it is usually four or five The injection should be given early in the morning, *otherwise the diuresis will disturb sleep* The effect is often enhanced by giving ammonium chloride gr 15 to gr 20, four times during the preceding twenty four hours, after meals The diuretic is also sometimes more effective if urea is given during the same day in doses of 120 to 240 grains, t d s, in a 40 per cent watery solution

Fluid can be removed from the body in other ways *Paracentesis*

of the abdomen accomplishes this very effectively if there is ascites, and also frees the downward movement of the diaphragm. *Paracentesis of the pleura* is especially important for a pleural effusion by collapsing lung tissue is a most serious embarrassment to a failing heart. All pleural effusions, except the smallest, should be removed as soon as possible in heart failure. If there is gross œdema of the legs and feet, fluid in quantity can be drained from them by *Southey's tubes* with great benefit. The patient should sit in a chair or a heart bed, with the legs lowered, for a few days previously in order to decant the greatest amount of fluid into the legs and feet. *Acupuncture* of the dorsum of the foot is probably as valuable a remedy as Southey's tubes for the treatment of gross and persistent cardiac œdema. The skin of the dorsum of both feet, in the metatarsal area is sterilised by soap and water and by the application of surgical spirit. Three or four small superficial nicks are then made into the skin on each side. Penicillin cream is smeared over the region. Sterile gauze is laid upon the cream. The feet and legs are left hanging downwards over a mackintosh sheet arranged so as to collect the fluid and deliver it into a large receptacle. The wounds are kept clean and covered with penicillin cream and a light sterile dressing, which is changed each day. As much as 30 litres of fluid can be collected in four to five days at the end of which time healing will start and rapidly be complete. A sodium free or sodium-low diet, associated with plenty of water by mouth has recently been introduced as a diuretic measure. It seems to act well in some patients by washing out electrolytes from the body and thus enabling the tissues to lose fluid.

Some symptoms call for particular consideration. Paroxysmal dyspnœa may need treatment as an emergency. The sudden attack of shortness of breath simulating spasmodic asthma usually occurs without warning, either at night-time, or in the day time as a result of some excitement or physical activity. The attack may be treated by various drugs, but the action of these is by no means certain. Theophyllin-ethylene-diamine (Cardophylin) can be given by intravenous injection. Morphia gr $\frac{1}{8}$ to $\frac{1}{4}$ combined with atropine gr 1/150 to 1/75, is frequently useful. *Oxygen administered by mask* may help. Glyceryl trinitrate gr 1/120 can be tried but is usually disappointing. Adrenaline m 5-10 of the 1 in 1000 solution subcutaneously is certainly helpful but there is some risk in its administration.

Dyspnœa and general distress is often much benefited by oxygen, in spite of the fact that in heart failure the circulation rate through

the lungs is lowered, so that more time is thus available for full oxygenation of the blood in the lung capillaries But much alveolar œdema is present, and by increasing the oxygen tension of the inspired air more of the gas can be driven through the thin layers of œdema fluid By the same process the plasma can be encouraged to take up some oxygen The result is that oxygen administration by tent mask, or double nasal catheter produces visible and great improvement in many cases *Venesection* is very helpful in those cases of heart failure with a clearly raised venous pressure, between ½ pint and 1½ pints of blood should be removed at a time, the best effect will be produced if this removal is done rapidly by drawing the blood under negative pressure through a large bore needle into a flask *Total thyroidectomy* is occasionally very useful in patients with chronic congestive failure in whom œdema remains and in whom deterioration seems to have ceased The choice of case for this treatment is a matter for expert assessment

Section III

THE MYOCARDIUM

CHAPTER VII

DIPHTHERITIC MYOCARDITIS

THE two most typical varieties of acute myocarditis are the diphtheritic and the rheumatic. In each case the heart-muscle fibres undergo hyaline and fatty degeneration, but these changes are more acute and more profound in diphtheria. Indeed in diphtheritic myocarditis the chief injury is that suffered by the heart-muscle fibres, whereas in acute rheumatic myocarditis, although some less degree of myocardial change occurs, most of the inflammatory change is found in the interstitial tissue of the heart.

In diphtheritic myocarditis the changes are produced by the exotoxin of the Klebs-Loeffler bacillus. The change in the heart muscle is an acute parenchymatous degeneration of the muscle fibre. Similar changes are seen, but to a less extent in the heart in pneumonia, influenza, typhoid fever, and certain other infections.

Parenchymatous diseases of the heart have three qualities, they are acute they are transient, and they terminate in either death or in complete recovery. Those few muscle fibres which may die in a patient who recovers, are compensated for by hypertrophy of the remaining fibres. The change in the case of diphtheria is rapid and profound, fatty degeneration being visible within six days from the onset in a severe case.

The functional changes in the heart muscle are as follows. Muscle tone is lost, the myocardium being somewhat dilated, the heart is enlarged and the action flabby. The power to meet sudden emergencies is diminished, so that a patient may die suddenly on sitting up or while emptying the bowels. The myocardium may become irritable, and damage to the bundle of His may occur. These changes show themselves by the presence of cardiac irregularities such as premature beats, paroxysmal tachycardia, flutter, and even fibrillation, or by the development of heart block. Electrocardiographically many patients who show no clinical abnormality are found to have a prolonged P–R interval, or even a bundle-branch lesion, indicating disease of the conducting tissues.

The physical **signs** are that the apex beat is somewhat displaced to the left, the heart rate is generally increased, the first sound develops a poor quality, is highly pitched and may be reduplicated; a systolic murmur may develop at the apex Congestive failure hardly ever occurs, for syncope or the development of irregularities usually prove fatal before it can develop Associated with the cardiac lesion there is also considerable peripheral circulatory failure or shock, shown by a fall in the blood pressure, sweating and pallor

The **treatment** of diphtheritic myocarditis is prophylactic If it is true that one might say of a vaccine dose, "Think of a number, halve it," so in talking of serum dosage one might say, "Think of a number, double it" Thus if it is thought that an initial vaccine dose should be 10 million, by giving 5 million a deleterious reaction is often avoided If it is proposed to give to a patient 15,000 units of anti-diphtheritic serum, by giving 30,000 units the risk of initial underdosage is greatly lessened Serum, in all appropriate conditions, must be given in large doses and as early as possible This provides the best chance of avoiding myocarditis in a case of diphtheria Whenever myocardial complications are feared 50,000 or even 100,000 units should be given on the first day the patient is seen Sugar by mouth by the rectum or intravenously is helpful to the inflamed heart, but any intravenous treatment must be small in bulk to avoid distending the weakened heart muscle If pulmonary œdema is present oxygen should be given in an efficient manner For an emergency nikethamide (0 2 to 0 5 gramme) intramuscularly or intravenously, or other analeptic preparations such as Methedrine (B W & Co), intramuscularly (15–30 mgm in 1 5 c c) or intravenously (10–20 mgm in 1 0 c c), are useful It is doubtful whether they have any direct action on the myocardium, but by stimulating the respiratory and vasomotor centres the systemic blood pressure is raised, the coronary circulation is improved, and the heart is benefited

The patient must be nursed throughout as flat in bed as is comfortable This resting period provides the only remedy, it is productive of an uninterrupted recovery if the causative poisoning has not been too intense, and if this has been removed or has disappeared Physical exertion during myocarditis is dangerous When the active inflammation has ceased four more weeks' rest in bed is usually essential

CHAPTER VIII

RHEUMATIC CARDITIS

A THOROUGH knowledge of acute rheumatism is of the greatest importance to a cardiologist, for it causes about 40 per cent of all cases of heart disease in the British Isles, and a high percentage in other countries, varying according to climatic conditions

Acute rheumatism is very dangerous It produces permanent defects early in life, the infection is recurrent no immunity develops during the disease, it cripples early and kills late Cure is therefore rare, and complete arrest is doubtful in a case where the heart is markedly involved The disease in children is chiefly cardiac and in adults is often restricted to the joints

That acute rheumatism is caused by an infection is proved by the following facts Fever is present leukocytosis is almost invariable and is proportional to the severity the joint effusions are inflammatory in type, the histology, both cardiac and synovial is that of an inflammatory lesion and the onset is very often accompanied by an acute tonsillitis hæmolytic streptococci are present in the throat

To understand the method of production of the signs and symptoms of acute rheumatism attention should be focused upon two separate aspects of the pathology One of these is the infection of the throat by streptococcus hæmolyticus The other is the allergic type of reaction observed in the body tissue An ordinary general streptococcal infection of the human body is one in which the organism enters possibly through the throat or through some wound, and proceeds to invade the blood stream and tissues generally The blood culture in such a case is often positive and organisms can be recovered from the urine during life and from the organs of the body post mortem But in acute rheumatism hæmolytic streptococci are present in the throat and they remain in this situation and exercise their influence at a distance upon the tissues The blood culture in acute rheumatism is always negative

An example of a simple allergic condition is that of serum sickness when the body has been rendered allergic to for instance horse serum Here a clinical picture is produced extremely like that of

acute rheumatism The medium-sized joints swell and become painful from an acute synovitis A skin eruption occurs, sometimes similar to that seen in some cases of acute rheumatism, and fever is produced An allergic reaction can of course be produced in most individuals experimentally, but allergic diseases seem usually to be restricted to those who are born with unduly sensitive tissues The same seems to be true of acute rheumatism The presence of hæmolytic streptococci in the throat initiates the rheumatic process only in susceptible individuals

If these two factors infection and allergy, are combined, an idea may be obtained as to the nature of the rheumatic infection The hæmolytic streptococcus plays a part analogous to that of an antigen in an asthmatic case That this is so is shown by the fact that skin tests reveal the presence of sensitivity to streptococcal proteins The effect of the sensitivity is to produce in the heart and joints the state of undue fragility and inability to stand up to the trauma of ordinary wear and tear so characteristic of the disease

Pathology

The pathological changes present in acute rheumatism provide much information concerning the method of production of the signs and symptoms of the disease and also offer the best key to the problems of diagnosis and treatment

The characteristic lesion of the disease is the Aschoff body, composed of a fibrous matrix, giant cells with several nuclei, fibroblasts and plasma cells and leukocytes which are chiefly lymphocytes This typical lesion is found in the synovial membrane of the joints, in the myocardium and endocardium and in the subcutaneous rheumatic nodules

The Aschoff body is probably evidence of a local inflammatory reaction to a particular type of toxic injury Three things may happen to Aschoff bodies, wherever they occur they may disappear, they may organise into fibrous tissue and thus cause valvular or other defects and they may even subsequently lead to calcification Having disappeared they may recur in the same or similar positions

The position of the Aschoff bodies, and of the rheumatic inflammatory lesion, is typical Trauma seems to be the factor which decides the progress of the rheumatic lesion, the joints are obviously vulnerable to trauma, and the subcutaneous nodules are nearly always at positions of friction Friction is an obvious factor in the production of pericarditis and endocarditis In the myocardium the nodules are chiefly found in close association with those collections

of arterioles, veins, and lymphatics which lie between the individual muscle-masses, where friction, tension, and pressure are also undoubtedly produced In the heart-valve cusps the vegetations are present only at the position of impact A final point to notice is that where complete rest is possible, as in the joints, recovery is always complete and rapid, so that the absence of trauma is a fundamental point influencing recovery

There is also seen in rheumatic carditis a secondary parenchymatous degeneration of the heart-muscle fibres analogous to that found in diphtheria, but it is considerably less severe

Clinical Characteristics

The typical attack of acute rheumatism is the attack of rheumatic fever, in which fever, sore throat, and profuse sweating are associated with an acute synovitis of one or more joints of the body The affected joints are swollen, slightly reddened, extremely tender and warm to the touch They are affected two or more at a time, but the severity of the synovitis varies greatly in the individual joints, so that the signs may subside partially or completely in one or more of the joints, only to appear in an equally severe form in others Some rather rare cases of considerable persistent fever without any obvious cause can also by shown to be rheumatic by their dramatic response to sodium salicylate The cardiac manifestations of acute rheumatism may show themselves in a variety of ways The most frequent is the development of myocarditis In addition pericarditis may occur and endocarditis is extremely common Since the myocarditis is associated with more or less endocarditis in nearly all cases, and with pericarditis in many, the term carditis, or pancarditis, is the label usually applied to cardiac rheumatism

Diagnosis

The diagnosis of rheumatic carditis is made by attention to the following points Certain symptoms and signs, in children especially, should always suggest acute rheumatism in the hitherto non-rheumatic, or activity in the rheumatic patient One of the most useful methods of obtaining rapid information as to quiescence or activity in rheumatic children attending a clinic, or an out-patient department, is the use of accurate scales The onset of rheumatic activity, even of a mild degree, is seen directly on a carefully kept weight chart, for the child immediately loses weight or fails to put on its usual normal increase Growing pains, whether they involve joints or muscles, must be considered rheumatic, particularly if the

arms as well as the legs are involved. Erythema circinatum or multiforme is frequently evidence of rheumatic activity, erythema nodosum is less often so.

Epistaxis has been regarded by some observers as a valuable symptom indicating activity, but seems to be less common in London children than in those of the United States. Tonsillitis is infrequently rheumatic, but in a rheumatic patient tonsillitis usually indicates an active rheumatism. Abdominal pain, with or without vomiting, is a common manifestation of acute rheumatism in children. It is possibly in some cases an indication of local rheumatic peritonitis. In children all rheumatic manifestations must be considered as indicating carditis until the contrary can be proved. Acute rheumatism also shows itself in the form of chorea.

Inflammation is shown by fever, tachycardia, leukocytosis, and a raised sedimentation rate.

Fever. Acute cardiac rheumatism is usually febrile. In some cases however the disease seems to make progress insidiously and in the absence of fever. Rheumatic nodules for instance, usually regarded as coexisting with active carditis in children, occasionally continue to appear without fever. But the presence of fever, otherwise not explained, must be regarded, in a rheumatic patient, as evidence of activity and therefore no febrile rheumatic patient should be allowed to undergo any avoidable exertion. A slight fever, often not exceeding 99° F. may continue for months, and may be the only or nearly the only, sign that the disease remains active. It is a risky step to ignore this low fever and to allow the patient to get up.

So important are slight variations of temperature and pulse rate that it is essential in cases of acute rheumatism to use adequate charts. Adequate charts are those in which the temperature, pulse, and respiration readings are actually all charted. Inadequate charts are those *in which, although the temperature* is *charted, the pulse* and respiration figures are recorded as such in a meaningless jumble at the bottom of the page.

The Pulse. The pulse rate is nearly always increased in the active stage of the disease, and the increase is usually in proportion to its severity. But in rheumatic children the heart is often hypersensitive to the stimulus of emotion, and a true reading may be hard to obtain. It is useful in these cases to record the sleeping pulse rate also. Other factors may be concerned in the tachycardia of acute rheumatism, or following it. Many children after complete

recovery, and while remaining well, seem to have abnormally excitable hearts, so that when seen in the out-patient department the rate is 120 or more This condition must be recognised and discounted

The Heart It is usually possible to deduce by a physical examination that in any given case the heart has been affected by rheumatic disease in the past In a severe case there is usually no doubt, in a mild but equally potentially dangerous case it may be most difficult to decide by examination of the heart that it is at the moment the subject of active rheumatic disease The heart is usually more or less enlarged, but often to only a slight degree Valvular lesions are the most obvious hall-marks of cardiac rheumatism, but they are the result of fibrosis, that is of the scarring produced by rheumatism months or years previously Attention must be focused upon changes actually being produced in the myocardium, and to a lesser extent in the valve cusps The force of the apical thrust diminishes in carditis The flabby apex beat is displaced to the left of its normal position, and may be difficult to define The first heart sound at the apex is an index of the vigour or the health of the heart muscle In acute rheumatism this sound loses force and becomes less pure in quality, according to the state of the myocardium In addition, other murmurs and adventitious sounds are sometimes of importance and suggest cardiac involvement Perhaps the most noteworthy point is definite variation in these sounds or murmurs from time to time in any one case For instance the development of an apical systolic murmur or of a localised systolic or diastolic sound suggesting soft friction is suggestive In some cases a distant diastolic sound is audible over the pulmonary area, like the murmur of aortic regurgitation, which disappears after a few days or weeks The cause of this sound is unknown, although it is well recognised

There may be evidence of endocarditis If mitral stenosis is present there is no doubt that the heart has been affected by rheumatism in the past Aortic regurgitation in a child or adolescent is nearly as diagnostic Mitral regurgitation if the physical signs are definite, is also highly suggestive In suspected rheumatism the signs of early mitral stenosis (p 135) must be differentiated with the greatest care from sounds and murmurs found in the normal young heart (Chapter XXXV)

The subcutaneous rheumatic nodules are characteristic They are found most usually over the elbows, the tendons of the extensors of the hands and feet, the knees, the dorsal spine, and over the

scalp They are fibrinous and rapidly disappear when the activity of the disease ceases They are most easily seen rather than felt, except in the case of the scalp The elbow, for instance, should be held in a good oblique light, *and the skin moved rapidly this way and that over the underlying bony joints* This will render it transparent in appearance and the nodules can very readily be seen They are slightly mobile under the easily moved skin Normal slight prominences are nearly always bilateral and equal in size The larger visible nodules are about the size of a dried pea, lesser nodules are common, and the smallest are scarcely larger than a *small pin s head Those on the scalp are two or three times larger than those present elsewhere, and can be easily felt through the hair* Nodules seem to be especially common in cases of acute rheumatic pericarditis

The joints in acute rheumatism are acutely inflamed, the degree of inflammation varying between slight and exceedingly severe They are swollen, sometimes a little reddened, painful, and warm to the touch The diagnosis of acute rheumatic synovitis can be confirmed by the therapeutic use of sodium salicylate This drug *should be given every two hours and in full doses, the usual amount* for a child being between 120 and 200 grains per day Double the quantity of sodium bicarbonate is generally prescribed with it Fifteen to twenty grains of sodium salicylate are administered every two hours, for owing to rapid excretion a high concentration would not otherwise be maintained in the blood In an uncomplicated case of acute rheumatism without carditis the temperature will almost always fall to normal or below within forty eight hours with *this treatment, and the joint symptoms will as rapidly disappear Should this not occur some other condition must be suspected, such* as acute osteomyelitis septicæmia or *malignant endocarditis* Should the temperature fall but not to normal, and should the joint symptoms disappear, the persistent lower temperature is strongly suggestive of the presence of a rheumatic carditis The absence of [illegible] indicates a diminution or a cessation of the rheumatic [illegible]

of help in determining whether the [illegible] *quiescent, but care must be taken to have sleeping pulse-rate figures* charted also in order not to be misled by *nervous tachycardia*

Blood Examination Three changes in the blood suggest activity anæmia, leucocytosis, and an increased sedimentation rate The anæmia is often not severe, the hæmoglobin being lowered to 65 or

70 per cent But in acute attacks, especially when acute pericarditis occurs, these figures are rapidly reduced further, even as low as 40 per cent The white blood count is generally increased in active cardiac rheumatism, and this increase may result from only slight activity The increase may be moderate in degree The usual total figure is 10,000 to 15,000 In pericarditis it may be as high as 25,000 Many observers find valuable a Schilling or Arneth count, in which the types of polymorphonuclear cell are differentiated In rheumatic activity there is a "shift to the left," or an increase in the bilobed young cells at the expense of the more mature

The sedimentation rate of the red blood cells is a simple and valuable means of estimating the activity of the disease The normal figure should be less than 10 mm in the first hour The sedimentation rate increases greatly with activity and falls rapidly with quiescence.

X-ray Examination Children confined to bed cannot be adequately examined by X-ray screening, but when portable radiological facilities are available comparison between films of the heart taken under comparable conditions may be helpful, revealing variations in size, small pericardial effusions, prominence of the pulmonary conus, and pulmonary changes, such as the so-called rheumatic pneumonia

Electrocardiography The electrocardiogram is of great use in assessing inflammatory changes, and the more extensively it is employed during the course of cases of acute rheumatism the greater is the number of deviations from the normal which it reveals These are transitory as a rule They comprise increase in the P–R interval, which is most common, greater degrees of heart block, which are more rare, changes in the shape of the QRS T complex such as slurring of the T wave, failure of R to reach the base-line, flattening of the T wave, and, in cases developing mitral stenosis, notching of and increase in the P wave, and right-axis deviation Irregularities such as premature beats, auricular or ventricular, may occur The importance of all these abnormalities is that they point to inflammation of the heart muscle

Treatment

There is no other disease in which more methods of treatment have been applied and with less success But, owing to the dangerous nature of rheumatic carditis and the frequency of its sequelæ, every rational therapeutic measure must be applied with the utmost thoroughness

The first point to be kept in mind is the relationship between injury and the position and degree of the rheumatic lesions The joints, the bony prominences and tendon sheaths, the heart valve cusp edges, the pericardial surface, the intermuscular areas in the myocardium, each and every one of these is a traumatised area

In health, inflammation is absent, or lubrication is effective, or tissue resistance is adequate But in acute rheumatism these factors are reversed and the tissues appear unduly friable Pain in the case of the joints acts as a protective agent, but the insensitive cardiac tissues continue relentlessly in action The rheumatic lesion in the joints heals, that of the cardiac tissues frequently does not, or does so only with local fibrosis The moral would seem to be that the inflamed heart must be guarded from everything that would increase its activity, until it is reasonably certain that all rheumatic inflammation has ceased If a patient in bed is allowed that degree of activity which will cause an average increase of five beats per minute throughout his waking hours, averaging twelve in number, his inflamed mitral cusp edges will be subject to 3,600 forcible impacts per day more than they would otherwise suffer It must be remembered that the force of every such impact on the aortic cusps will be greater than the diastolic pressure, which in the case of a child will exceed 40 millimetres of mercury The impact upon the mitral cusps is also considerable Thus *rest* must be insisted upon, and the child should be kept in bed until there is no evidence of activity of the disease as shown by fever, tachycardia, myocardial inflammation, as determined by changes in the electrocardiogram until the anæmia which may have been present has disappeared, until the leukocytosis has gone, and until the sedimentation rate has fallen to normal This period of rest in bed may be as long as from three to nine months A further period of rest of from three to six weeks should be added in a severe case, as a precautionary measure

Climate appears to have an effect upon the prevalence and the activity of acute rheumatism, as it has upon the incidence of scarlet fever Some New York children suffering from active rheumatism were transferred to a convalescent home in sub-tropical America where they rapidly lost all signs and symptoms of activity Whether this change can be put down to sunshine, fresh air, good food, or a persistently warm climate is impossible to say When, however, they were returned to New York many of them very rapidly suffered from recrudescence of the disease

In Brazil, the coastal tropical areas are practically free from this disease, whereas the more temperate areas in high altitude and within

easy travelling distance have a moderately high incidence of acute rheumatism and scarlet fever It would be advisable theoretically to nurse a case of acute rheumatism, and even to educate the child subsequently, in such a relatively immune climate

Diet has no noticeable effect upon the course of the disease It has been found that a diet rich in vitamins has no effect upon the development of acute rheumatism A properly balanced diet is therefore all that is required

Drugs Sodium salicylate has naturally been used freely in rheumatic carditis, since there is no doubt of its efficacy in the acute articular form of the disease But unfortunately its therapeutic action upon the heart is exceedingly slight, if it occurs at all However it is a reasonable practice always to give it in full doses as already described, provided that the patient tolerates it without difficulty Unless the drug is prescribed with twice the quantity of sodium bicarbonate, symptoms of intolerance are apt to occur For the sodium salicylate reacts with the hydrochloric acid of the stomach to form sodium chloride and salicylic acid This latter substance is a gastric irritant and causes vomiting The salicylate dosage is at first in the neighbourhood of 180 grains per day varying with the size of the child and its tolerance to the drug and it is continued at this level so long as fever persists The dose can then be halved and ultimately stopped when all signs of active disease have gone Iron as ferrous sulphate should be given for the anæmia

Cardiac complications may need individual treatment Auricular fibrillation, although uncommon, will require digitalis in sufficient dosage to reduce the apical heart rate to a reasonable figure which will depend upon the degree of fever also present Congestive failure will require treatment by diuretics

Prophylactic Treatment

Prophylaxis has been tried by attempting to immunise children against acute rheumatism, using hæmolytic streptococcus filtrate But the results on the whole have been disappointing *Sulphonamide* has been given over a long period and in small doses with the idea of discouraging streptococcal invasion in rheumatic children No toxic, hæmic, or cutaneous reactions of any severity are usually observed The daily dose is 0 5 gm to 1 2 gm Hæmolytic streptococci are less often obtained from the throat cases so treated than from those of controls While taking sulphonamide patients *are said to be less liable to a major attack of acute rheumatic fever* It must be remembered, however, that there are patients who take

sulphonamide less satisfactorily, and that in an active phase of the disease the drug may tend to cause an exacerbation Penicillin lozenges should be given if signs of activity are noticed

The Tonsils A final therapeutic point concerns the tonsils Tonsillectomy during rheumatic activity can, and occasionally does, cause a definite serious and acute flare-up of the disease, such as an attack of pericarditis The operation is, however, safe during periods of quiescence The rule should be that definitely infected tonsils should be removed, but that the time of their removal should be carefully chosen Sinusitis is frequent in rheumatic children, and treatment of this is essential Nasopharyngeal infections must be carefully treated and nursed Local treatment, by oily or other spray, should be used when necessary The full cooperation of a good conservatively minded nasopharyngeal surgeon is often helpful

The **Prognosis** is by no means easy Generally the younger the patient the more seriously is the heart affected, so that one might say that a first attack of acute rheumatism under the age of fifteen indicates a poor outlook, and over the age of twenty-five a reasonably good one The younger the child the more serious is the outlook, and the seriousness is also proportional to the number of attacks of acute rheumatism suffered It is impossible to foretell during an attack whether the child will be left with a permanent endocarditis, fibrosis of the heart valve probably takes at least six months to develop, so that mitral stenosis is never a sign of activity if the murmur is heard during an attack Before giving an opinion as to whether mitral stenosis is likely to develop from the attack under observation it is wise to wait for six to twelve months and examine the child again The presence of subcutaneous nodules is generally thought to be an indication of a severe attack

CHAPTER IX

MYOCARDIAL DEGENERATION

MYOCARDIAL degeneration is a term which has been used more extensively of late years to describe changes in the heart muscle These changes may be localised in sizeable patches visible to the naked eye, or may be microscopical and diffusely scattered in the muscle The origin of the change is probably in most cases vascular, and secondary to arterial disease

The manner in which myocardial degeneration shows itself varies enormously, and depends entirely upon the extent of the lesion and its anatomical position in the heart muscle It may cause heart failure if a sufficient mass of muscle fibres is incapacitated but failure is absent when the lesion is small It also may cause disorders of conduction if the bundle of His is picked out, or other forms of irregularity if the disease is restricted to the auricles or to a local area in the ventricular muscle

For this reason its clinical manifestations are usually described separately and they are scattered widely in textbooks under the appropriate headings, while the underlying lesion although so universal is left in comparative obscurity

The manifestations of myocardial degeneration depend upon the area of the heart affected by the process, and by the type of lesion present Thus in many cases coronary disease forces attention upon the symptoms of angina of effort or of cardiac infarction In others auricular fibrillation, due to auricular myocardial degeneration is thought of solely in terms of the irregularity, which in this case is particularly responsive to digitalis therapy Again, in complete heart block the bradycardia of thirty beats per minute or possibly the sensational Stokes-Adams attacks monopolise the stage Occasionally, however, patients undergo progressive cardiac failure, ultimately congestive in type, without any such striking symptoms or signs, and the label myocardial degeneration is often restricted, rather unjustifiably, to this group

The essential cause of the condition, in the majority of cases, seems to be cardiac arteriosclerosis If a large vessel or a large branch is involved by atheroma, the lesion may be so localised that the

injured heart muscle heals and is compensated for by hypertrophy of the rest of the heart If, however a large number of terminal arterioles are affected, the chance of a collateral circulation developing becomes lessened or impossible, and the result is gradual and inevitable deterioration

Not only does myocardial degeneration occur as the sole abnormality it also is a frequent complicating factor in other forms of chronic heart disease Most patients with chronic rheumatic carditis who survive to the age of fifty or more suffer in addition so far as the heart muscle is concerned from local arterial changes with subsequent degeneration This is true also of patients in whom chronic syphilitic aortitis affects the mouths of the coronary arteries Thyrotoxicosis also in elderly subjects is not infrequently complicated, as regards the cardiac vascular system by myocardial degeneration of vascular origin A final point is that the so called hypertensive heart is due to myocardial degeneration complicated by the additional great handicap of a high blood pressure

When the symptoms of myocardial degeneration are those of *chronic cardiac failure they require no further elaboration here* (see p 31)

Signs

The heart is usually increased in size especially the left ventricle from dilatation and some degree of hypertrophy The cardiac impulse is frequently rather diffuse and X ray examination confirms both the general enlargement and the rather boot shaped appearance of the enlarged heart

The first sound is apt to be poor in quality and may be reduplicated A systolic murmur is sometimes present at the apex possibly a result of stretching of the mitral ring A systolic murmur may also be heard at the aortic base conducted into the vessels of the neck, and due to the atheroma and irregularity of the aortic valve This aortic systolic murmur is in a few cases referred backwards to the apex where it is sufficiently loud to cause difficulty in diagnosis The aortic second sound is often increased, even with a normal blood pressure the accentuation being due to the proximity of the atheromatous ascending aorta to the chest wall on the right side

Certain other vascular signs are common in these patients The aorta may be remarkably opaque on X-ray screening, from the presence of atheromatous degeneration, and it is often tortuous The retinæ may show some tortuosity of the arterioles The peripheral arteries may be thickened and winding Even in the absence of

hypertension the diastolic blood pressure may be increased giving a reduced pulse pressure of, for example 136 108

The electrocardiogram will, of course, show the presence of the more remarkable or sensational manifestations referred to above if they are present, such as evidence of coronary infarction, auricular fibrillation, heart block, or bundle-branch block, etc But apart from these, and in an uncomplicated case left axis deviation is frequent The T wave may be flattened, diphasic, or inverted in Leads 1, 2, and 4 There may be some slight S-T deviation Premature beats may be unusually frequent and increased by exertion

CHAPTER X

THE THYROID GLAND AND HEART DISEASE

THE heart can be affected either by an over secretion or by an under-secretion of thyroxin The over secretion may be the result of disease or of oral administration The under-secretion may be due to congenital or to acquired disease

The normal heart affected by hyperthyroidism beats more rapidly and with a greater output, and may develop premature beats I have seen auricular fibrillation develop after the oral administration of thyroxin The indiv [illegible] occurrence, and remaine [illegible] was normal clinically, [illegible] before and after the attack which lasted three days Thyroxin administration causes an increased pulse rate increased pulse pressure increased myocardial metabolism, and all other manifestations of thyrotoxicosis with the exception of exophthalmos

GRAVES'S DISEASE

Three problems in relation to thyrotoxicosis may confront the cardiologist *The first is that in manifest Graves's disease, the* cardiac condition is clinically the most important The operative risk is cardiovascular, and the chief efforts of treatment are directed to the elimination of that risk for most of the serious cases ultimately require partial thyroidectomy Whether thiouracil will radically change this opinion is as yet not decided

The second and third problems are primarily diagnostic for the symptoms and signs of thyrotoxicosis may in each case be obscured by a cardiac clinical picture The first of these is early or mild hyperthyroidism in young people, which has many features in common with effort syndrome or neuro circulatory asthenia The second is *unexplained auricular fibrillation, with or without congestive failure,* in middle-aged people

In reference to the first example both thyrotoxicosis and effort syndrome manifest lassitude, palpitation, sweating, tremor, and some loss of weight, and the differential diagnosis is frequently difficult,

more especially because the basal metabolic rate figure in effort syndrome cases may be vitiated by inability of the patient to relax and by general nervousness In effort syndrome, however, the palpitation is much exaggerated by emotion, the sweating is localised to axillæ, hands, or forehead, and the sweat is a "cold sweat", the tremor is often coarse, and left-chest pain of a functional nature is common

In thyrotoxicosis it must always be remembered that the body tissues are oxydised more quickly by the faster metabolism, and therefore generate and lose heat faster, so that symptoms and signs consequent upon this are constantly present Weight is usually lost steadily, tachycardia persists to some degree day and night and a record of the sleeping pulse rate is helpful, the skin is uniformly warm to the patient and warm to the touch, and is moist all over without exaggerated local sweating, so that the patient no longer feels the cold, but becomes over-sensitive to heat, tremor is fine These are the most reliable clinical diagnostic points in all cases of suspected thyrotoxicosis There is in most of these early thyrotoxic cases a small amount of exophthalmos, which appears either as a rather staring expression, or as a slight prominence of one eye as compared with the other Also there is usually some enlargement of the thyroid gland, even though this be slight

In the second group, where diagnosis is difficult, similar symptoms and signs must be looked for, here the typical case is an elderly patient with auricular fibrillation, in whom is found no obvious cause for this irregularity, such as mitral stenosis or arterial disease

When the diagnosis is in doubt the basal metabolic rate should be *determined* *In either type of case iodine administration may* provide a valuable clinical test The heart rate is charted for some days When its level is steady, Lugol's iodine is given in doses of 3 5 minims t d s If there is thyrotoxicosis a demonstratable and durable fall in the heart rate occurs after a few days, and simultaneously the thyroid gland, hitherto soft or impalpable, may harden If a reliable basal metabolism apparatus and a trustworthy technician are both available, and if the patient's full cooperation can be obtained, the basal metabolic figures constitute a most valuable help to diagnosis and progress But these three variables are frequently sufficiently uncertain to jeopardise the accuracy of the test Moreover congestive heart failure may increase the basal metabolic rate by 10–15 per cent in the absence of thyrotoxicosis

In all cases of Graves's disease thyroxin produces both a persistent overdriving of the heart and a toxæmia The manner of causation

of the symptoms and signs in the cardiovascular system is briefly as follows Hyperthyroidism, or dysthyroidism, results in symptoms of sympathetic stimulation one manifestation of which is the increased basal metabolic rate The tissues therefore need more oxygen the circulation rate is much accelerated, and tachycardia is produced, ultimately some generalised cardiac hypertrophy also develops At the same time the excessive thyroid secretion seems to have a direct action toxic or metabolic, on the heart muscle, and this becomes dilated to a greater or less extent, and sooner or later shows signs of failure A secondary effect of this action upon the heart muscle is an increased excitability, manifested by a tendency to premature beats, auricular fibrillation, and, more rarely, auricular paroxysmal tachycardia or flutter

The effect of thyroid overaction upon the abnormal heart is also a matter of great clinical importance If rheumatic disease is already present symptoms of cardiac failure are accelerated, and in elderly people myocardial degeneration, from arteriosclerosis, is more likely to be increased Thyrotoxicosis is also apt to speed up the development of hypertension where this is latent or still in an early stage

The degree of failure in thyrotoxicosis varies greatly in different cases It tends to be more severe in the secondary type of Graves's disease or toxic adenoma of the thyroid as it is sometimes called But this is not always true and no useful therapeutic purpose is served by regarding primary and secondary Graves's disease as two distinct entities

The fundamental basis of therapy is the same in all cases The first step in successful treatment is careful evaluation of the degree of failure In slight cases tachycardia is the only sign and palpitation and dyspnœa the only symptoms present In more severe cases the heart is enlarged the degree of enlargement being proportional to the degree of failure In the most severe cases auricular fibrillation is generally present This irregularity is not only symptomatic, it adds mechanically to the severity of heart failure When the source of the thyrotoxicosis has been removed by treatment, the cardiac insufficiency disappears in a remarkable way Surgical treatment is still the best means of removing the source of poisoning in a severe case X-ray treatment is less satisfactory owing to the greater difficulty of estimating exactly how much of the gland is being destroyed In mild cases it is a valuable method of treatment Thiouracil has proved its value as a temporary alleviator of the disease Its place as a curative agent is still undefined Should thyroidectomy be decided upon, the medical preparation of the

patient becomes of the utmost importance Only if this is seriously and carefully undertaken can the mechanical removal of the gland be carried out with the minimum of risk

Treatment

From the medical or preparatory point of view the treatment of thyrotoxic heart disease falls under four main heads These are rest, diet the use of iodine or of thiouracil and in cases with auricular fibrillation of digitalis Treatment by these methods should be so synchronised that the optimum results to be expected from each will be obtained simultaneously For it is clear that relief to the patient will be at its maximum if treatment by each method can be so managed that its greatest effect will coincide with the greatest effect produced by each of the other methods The necessity for thus carefully planning the treatment will be clearly understood when it is remembered that treatment with iodine if the usual full doses are given leads to a marked improvement in patients with hyperthyroidism in about fourteen days and that after this time although the drug be continued in the same dosage a partial relapse occurs On the other hand rest and diet may have to be persisted in for several or for many weeks longer before full benefit is seen Again digitalis takes a variable time to yield its maximum effect Therefore the best results can be obtained only if iodine and digitalis are administered in such a way as to secure the most satisfactory therapeutic response at the time when rest and diet shall have done their utmost in improving the patient's condition Not until this has been accomplished is the patient ready for thyroidectomy

If the case is mild or of moderate severity the above programme will suffice In more severe cases especially where advanced myocardial disease is shown by the presence of gross cardiac enlargement often with auricular fibrillation thiouracil may be useful as an emergency measure because of its effect of diminishing thyroid activity within about two weeks But it simultaneously produces so gross a vascularity of the gland that surgical removal becomes temporarily difficult In such a situation where the state of the heart has demanded rapid alleviation and where thiouracil has been given successfully the improvement can be maintained after stopping the drug by the immediate exhibition of Lugol's iodine in full dosage for two weeks At the end of this time the vascularity will have diminished sufficiently to allow of surgical treatment Close co operation with a surgical colleague with frank interchange of views and information is essential in treating these really ill and difficult

patients The selection of the right type of operation, whether ligature of arteries only, or partial removal of one lobe, or bilateral partial thyroidectomy, will necessitate very careful consultation between physician and surgeon

The commencing dose of thiouracil for a normal adult is 0 6 to 1 0 gramme daily, in divided doses The maintenance dose is about 0 2 gramme daily for an adult During *treatment* the white blood cells must be counted every two days for agranulocytosis can occur Some leucopenia is usual but when the bone marrow becomes dangerously poisoned this preliminary fall is then followed by a further progressive diminution This situation must be met by withholding the drug by transfusion of packed cells, by pentnucleotide injections and by large doses of ascorbic acid (500 mg daily) Packed cells are given rather than whole blood so as not to overload the heart

Rest in Bed This must be complete The patient should be isolated from all domestic financial or business worries A quiet room must be chosen and visitors should be strictly limited in numbers A bedside telephone is not permitted Wireless addicts are silenced To assist mental rest sedative drug treatment is wise in severe cases sodium phenobarbitone ½ 1 grain twice or three times a day during the stage of more severe nervous excitement is probably the most efficacious The services of a trained nurse are essential in all pre operative cases The patient should be kept strictly in bed during the whole course of medical treatment The length of time which this requires averages about six weeks but may vary between four weeks and three months

Diet A high calorie value is essential and the food should consist of normal solid meals The calorie value of the daily diet should be between 3 000 and 3 500 and the patient should be encouraged to eat well The meals should consist, as far as possible, of food to which the patient is accustomed and should include in addition three pints of milk taken as such or in junket, cocoa coffee, Horlicks or Ovaltine Two ounces of cream and four to six ounces of sugar should also be added The effect of the high calorie diet, together with the rest in bed, should be to enable the patient, first to make good the wastage caused by the increased basal metabolic rate and secondly, to put on weight

Iodine Therapy Iodine should be prescribed with great care, particular notice being taken of its hardening effect on the thyroid gland The maximal hardening of the gland can be obtained within a relatively short time (about fourteen days) if full doses of iodine

are given, but when this point has been reached the drug appears to be without further avail, and the patient once more loses ground Iodine, therefore, should be taken in smaller doses, which are enough to cause the gland to become fairly firm It is kept in this state until the general condition of the patient has sufficiently improved to warrant operation Three or four days before the date fixed upon for thyroidectomy full doses of iodine are administered

The dose at the beginning of treatment is 2 to 5 minims of Lugol s solution once to three times a day in milk As soon as the gland starts to become somewhat firm the dose is reduced to 1 to 2 minims twice or three times a day enough being given in any particular case to keep the gland firm and elastic, but not hard This dose is persisted with until three to five days before operation, when it is increased to 10 minims three times daily after food In primary Graves's disease (primary toxic goitre) the true consistency of the gland is readily determinable In secondary toxic goitre (toxic adenoma of the thyroid), where hard nodules exist already in the gland, palpatory information is not so easy to assess in these cases attention should be directed to the feel of the gland away from the hard adenomatous areas An alternative method is to concentrate attention upon the other methods of treatment omitting iodine until seven to ten days before operation, when it is given in doses of m 10 to 15 t d s, p c But here also operative treatment must wait until the patient is improving in weight and in general condition

Digitalis Therapy Digitalis is useful only in cases with auricular fibrillation Employment of this drug in cases of thyrotoxic tachycardia with normal rhythm is a waste of time This statement applies to all preparations of digitalis and strophanthus both pharmacopœial and proprietary In the presence of auricular fibrillation digitalis may be used The best results are obtained from it when œdema of the feet and other signs of severe congestive failure are present In cases not thus complicated it will probably be found that rest, diet, and iodine, as described above will slow the heart rate more than will digitalis Patients with fibrillation of the auricles but with no signs of failure should be given 40 minims of tinct digitalis or 4 grains of digitalis folia (B D H) a day for three days before operation Patients with auricular fibrillation and signs of congestive failure should receive 40 to 60 minims of tinct digitalis, or 4 to 6 grains of digitalis folia a day for four to six days at the commencement of the period of medical treatment If symptoms of anorexia or nausea appear, the dose is reduced or temporarily omitted, in the absence of such symptoms of overdose the drug is continued in

reasonable doses as long as signs of failure persist When severe and protracted attacks of vomiting due to some cause other than digitalis complicate the heart failure the drug may be administered per rectum, three doses being given at four-hourly intervals A maintenance dose would be 60 minims in water twice or three times a day

The heart rate is raised in thyrotoxicosis when normal rhythm is present When fibrillation occurs, and is treated with digitalis, no attempt should be made to slow the ventricular rate below the figure which would be expected if normal rhythm were present in a patient with that degree of thyrotoxicosis Thus an apical figure of 100 per minute, for example, should be the therapeutic target, not 72 per minute When congestive failure with œdema is present the mercurial diuretics should be given

Thyroidectomy The medical treatment outlined above will lead in most cases to a diminution in the signs of hyperthyroidism, an increase in weight, and a fall in the heart rate So long as improvement continues as judged by these criteria, medical treatment should be persisted in When the improvement becomes considerably less rapid, or when it appears that further improvement is not to be obtained by these measures, surgery should be considered, and is in most cases necessary Thyroidectomy is contraindicated, as a rule, if weight is being continually lost, if the heart rate is above 120, and if there are signs of gross failure To operate on a thyrotoxic patient whose weight is falling is to invite disaster It is analogous to sailing a boat over sandbanks on a lee shore and on a falling tide Exceptions to these rules are occasionally met with, but judgement as to the safety of operation in them is a skilled matter, and the risk is great Should severe symptoms persist ligation of the thyroid arteries may have to be carried out as a preliminary to thyroidectomy

Post-operative Failure Post-operative acute failure is due to a sudden liberation of thyroxin by manipulation of the thyroid, and to its absorption from the field of operation It is treated by the following measures

(1) Continuous oxygen administration, by oxygen tent, mask or nasal catheter

(2) Continuous administration, per rectum or intravenously, of saline containing 5 per cent glucose

(3) Sugar should also be taken by mouth, and insulin in the proportion of one unit for every 2 grams of sugar ingested should be injected intramuscularly

(4) Sodium iodide, 20 to 40 grains in 10 c m of sterile water, should be given intravenously twice daily

(5) Lugol s iodine should be prescribed in milk in doses of 30 minims four-hourly, or per rectum in doses of one drachm four-hourly

Treatment of Auricular Fibrillation A high percentage of patients who before operation had auricular fibrillation lose the irregularity spontaneously after thyroidectomy Should this not occur within three or four weeks it is desirable to restore the normal rhythm by means of quinidine sulphate (p 99) But if a total of 30 to 40 grains of quinidine has not produced the desired effect it is wiser to desist, unless the physician has seen a sufficient number of cases to feel confident that a further increase in dose in the particular case is a safe measure Signs indicating that a cessation of quinidine treatment is desirable are buzzing in the ears and deafness, feelings of faintness or dizziness, and any considerable rise in the ventricular rate

No case of thyrotoxic heart disease with auricular fibrillation should be under the care of a nurse who cannot accurately count the heart rate at the apex with a stethoscope If the heart rates recorded on the chart cannot be relied upon by the physician, he is not in a position to estimate with full accuracy the clinical position This is equally true whether he is treating the auricular fibrillation with digitalis or with quinidine

Prognosis

After thyroidectomy the cardiac insufficiency disappears in most cases in a remarkable manner It is the author's experience that the long term prognosis depends upon whether any disease additional to the thyrotoxicosis, such as rheumatism or arteriosclerosis has affected the heart In a careful follow-up of patients after partial thyroidectomy, at St Bartholomew's Hospital, it was found that they fell into two groups those who still had evidence of heart disease, although the hyperthyroidism had been cured by thyroidectomy, and those with normal hearts In the former group nearly every individual presented clinical evidence of rheumatic or arteriosclerotic heart disease In the latter group no evidence of heart disease was discovered It would seem that the permanence *of any cardiac incapacity after hyperthyroidism and thyroidectomy* depends chiefly upon whether the patient's heart has been previously diseased Transient thyrotoxicosis may accentuate and may perpetuate the effects of rheumatic or degenerative cardiac disease, but it does not permanently damage a normal heart

Another abnormality which tends to follow thyrotoxicosis is hypertension This is not present in all patients who have suffered and recovered from Graves s disease but the incidence is certainly higher than normal among such cases The condition may result either directly from the thyrotoxæmia or from the wear and tear

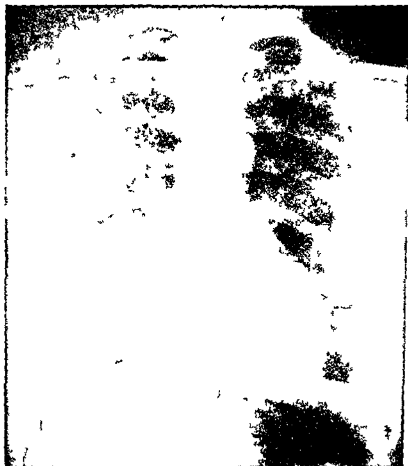


Fig 10 —Myxœdema

Transverse card ac diameter 13 5 cm There is g neral enlargement of the heart previous to treatment by thyrox n

produced by the increased metabolic rate over a period of years It is my opinion that the latter is the more probable hypothesis and that the thyrotoxicosis accelerates the onset of a condition which would in any event have occurred later on

MYXŒDEMA

The diagnosis of myxœdema depends upon certain classical symptoms and signs The symptoms are sleepiness lethargy

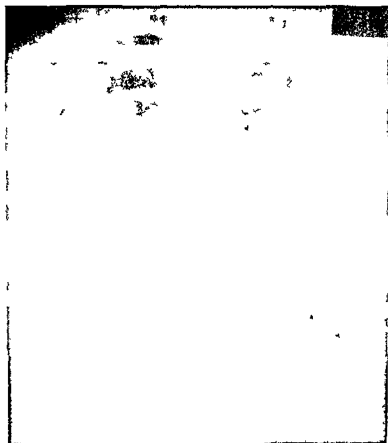


FIG 11—MYXŒDEMA SAME PATIENT AS FIG 10
After treatment with thyroxin for five months The transverse cardiac diameter has decreased to 11 cm

sensitivity to cold diminution in the powers of concentration and of rapid cerebration The signs are a change in facial appearance shown by increased coarseness and puffiness of features and loss of hair and eyebrows increase in weight bradycardia and dryness of

the skin Hoarseness or huskiness of the voice, probably due to some laryngeal thickening, is not unusual

The heart in myxœdema is often abnormal, but the gross physical changes may be present without dyspnœa or other evidence of abnormal function

The chief signs are enlargement of the heart, bradycardia, some increase in the blood pressure, and characteristic abnormalities of the electrocardiogram In some cases auricular fibrillation may occur Heart failure, slight or severe may supervene

It is interesting that both excess and deficiency of thyroxin may produce analogous effects As in thyrotoxicosis, so in myxœdema, if cardiac disease due to some other cause is present, or if there is slight hypertension, these abnormalities become accentuated When angina of effort occurs in individuals without myxœdema the pain is accentuated by the administration of thyroxin, and in such patients it can also be relieved by thyroidectomy, but angina of effort, if it occurs in patients with myxœdema, is relieved when the condition is rectified by giving thyroid extract The heart, in thyroid disease of either type, lacks the optimum amount of thyroxin and gets too much or too little

The enlargement of the heart shadow in myxœdema is considerable (Figs 10 and 11) For instance, the transverse diameter, by orthodiagram may measure 14 0 cm , after adequate treatment the figure may diminish to 12 0 cm It is uncertain whether this enlargement is due to pericardial fluid, or to an actual œdematous condition of the muscle

The electrocardiogram shows a low voltage curve, with flattened or inverted T waves in Leads 1 and 2

All these changes disappear as a result of adequate thyroid administration, and the physical efficiency of the heart returns to normal Should this fail to occur it is probable that any residual abnormality is due to some additional cardiac disease, which must be considered separately The prognosis must be modified accordingly

CHAPTER XI

THE HEART IN ANÆMIA

An adequate supply of oxygen is an absolute necessity if the heart muscle is to remain healthy There may be a deficiency in local supply of blood through the coronary circulation, followed by local myocardial degeneration, and subsequently local cardiac failure or scarring with more or less recovery of function Sometimes the blood itself is deficient in hæmoglobin, and then the heart is affected as a whole, according to the rapidity of the onset of such an anæmia, the failure of the heart is rapid or slow The changes produced by anæmia are frequently very severe, so that in a case of pernicious anæmia the post-mortem appearance presents an extreme example of widespread fatty degeneration of the majority of the heart-muscle fibres Such extreme degrees of change are not reversible, but recovery from minor degrees of hyaline degeneration, or possibly of fatty degeneration, may take place Such recovery if it occurs is in time complete

Myocardial symptoms and signs may follow any type of anæmia provided it is sufficiently severe Pernicious anæmia, microcytic hypochromic anæmia, or the anæmias following severe loss of blood from peptic ulcer or from uterine fibroid, can produce definite cardiac symptoms and signs

In addition, the lowered viscosity of the blood itself may give rise to certain manifestations A sufficient diminution in the total number of red blood cells produces a watery and less viscous blood, and this changes the physical resistance offered to the circulating fluid by the chambers and valves of the heart and by the peripheral arterioles This no doubt is responsible to a large extent for the so-called hæmic murmurs and also for the diminution of the diastolic blood pressure which is not uncommon in anæmia

Since the hæmoglobin is diminished it is clear that, in order to maintain the nutrition of the tissues of the body, an increased circulation rate becomes necessary This is a further factor responsible for the cardiovascular manifestations of anæmia

Symptoms

The usual symptoms characteristic of the type of anæmia in question, and of its cause, must of course be added to the cardio-

vascular picture Lassitude, headache, giddiness, faintness or fainting, or gastric disturbances, are present in most cases

The chief cardiac symptoms are shortness of breath, which is evidence of a diminished circulatory efficiency, and palpitation Palpitation is more prominent in anæmia than it is in most types of organic heart disease, the reason no doubt being the increased vigour of cardiac effort needed to maintain the increased circulation rate Moreover, some degree of anæmia nearly always causes hyperexcitability of the nervous system, so that this increased cardiac effort is gradually noticed by the patient

Pain is another cardiac symptom not uncommon in all types of anæmia It may be of the left submammary type found in functional disorders, or it may belong to the classical angina of effort group In this case it is central, sometimes radiating to the arm or arms and directly proportional to the degree of exercise being taken by the patient at the time (see p 209) Angina of effort is a well-known manifestation of pernicious anæmia, but it may occur also in microcytic anæmia and occasionally follows the anæmia produced by peptic ulcer With the successful treatment of the anæmia this pain, although of the coronary type, may completely disappear and remain in abeyance

The picture of general congestive failure is not very marked, and shortness of breath and orthopnœa are less obvious than in patients with left-sided heart failure, the reason being that in anæmia there is no increased pulmonary congestion The classical sign of anæmia is pallor, which must be critically analysed, for the facial appearance is frequently misleading, from the use of cosmetics, and is also often equally misleading in patients who, because of an excessive degree of peripheral vascular spasm, look pale although their hæmoglobin may be 100 per cent

Inspection of the neck and thorax shows an increased pulsation, both arterial and venous, due to the increased circulation rate, and also reveals some increased forcefulness of the pulsation of the heart itself Palpation however will not detect the strong thrust suggested by inspection The apex beat may be somewhat to the left from cardiac hypertrophy and dilatation On auscultation the first sound is frequently impure and accentuated Murmurs of the so-called hæmic type are audible The most usual of these are a systolic murmur at the apex, and a systolic murmur at the pulmonary base

Besides causing murmurs in previously normal hearts anæmia may produce myocardial stretching and thus murmurs of organic valvular regurgitation For instance, in a patient whose left ventricle

has become considerably dilated, a systolic murmur at the apex conducted into the axilla, typical of mitral regurgitation, may be present A much rarer organic murmur caused by anæmia is the murmur of aortic regurgitation, due to dilatation of the aortic ring Anæmia may greatly modify the murmurs produced by an already existing organic disease The patient may have a hæmoglobin of 50 per cent , and in addition typical mitral disease, with well-defined presystolic thrill and murmur, and loud systolic murmur If the anæmia is successfully treated it is found that the physical signs of mitral stenosis disappear, leaving only the systolic apical murmur A similar change may be seen, for instance, in a man with a systolic murmur of mitral regurgitation at the apex and a loud murmur of aortic regurgitation down the left border of the sternum In such cases successful treatment of a severe anæmia may cause the murmur of aortic regurgitation to become nearly inaudible Whether these changes are due to an increase in the viscosity in the blood or to an improved myocardial tone is uncertain Treatment of the anæmia may also reduce the size of the heart

The pulse in anæmic patients is not infrequently of the diacrotic variety, a well-marked diacrotic notch being easily palpable In addition the blood prsssure in such patients is often lower than the average, and treatment of the anæmia will increase the pressure by 10 or 15 mm of mercury, so that it once more becomes normal

The electrocardiogram in severe anæmia shows certain abnormalities which usually disappear with the disappearance of the anæmia The P–R interval may be increased, from impairment of function of the bundle of His The curve as a whole may have a lowered voltage There may be some R–T elevation or depression, the T waves may be diphasic or flattened, returning to normal with the blood picture The X-ray may show some enlargement of the heart as a whole, which disappears as the patient regains a normal blood picture

Treatment will remove completely and permanently the cardiac symptoms and the cardiac signs due to anæmia

The **prognosis** is, therefore, good Even in a patient with angina of effort from pernicious anæmia the pain usually completely disappears, and the patient may remain quite well for years

Section IV

CARDIAC DISORDERS AND IRREGULARITIES

CHAPTER XII

PREMATURE BEATS

The term premature beat is preferable to that of extrasystole for such contractions are nearly always premature in point of time. The ventricular variety does not cause any actual increase in the total number of heart beats per minute, and the auricular variety a small increase only, although both are ectopic, that is, arising away from the normal origin. They come from some focus in the muscle,



Fig. 12

auricular ventricular, or nodal, which succeeds in producing an excitation wave before the next normal beat is due.

Fig. 12 shows first three normal cardiac cycles, then a premature ventricular beat, then, after two normal cycles, a premature auricular beat.

The fourth normal auricular contraction transmits to the ventricle an impulse which reaches it when the premature ventricular beat has made the chamber refractory, so that no corresponding ventricular contraction follows. It is clear that the interval between the two normal, but interrupted, heart cycles is equal to two normal heart beats. In other words, the dominant rhythm is not interrupted. But the premature auricular beat transmits its premature impulse to the ventricle, which also therefore contracts prematurely. Thus the interval between the normal, but interrupted, heart cycles is here less than two normal heart beats.

It is easy from Fig. 12, and from the knowledge that the premature beats are ectopic, to construct the electrocardiogram. In the case

of the ventricular type the abnormal beat arises somewhere in the ventricular muscle. Since it travels at first through ordinary ventricular muscle, and only later through the special conducting system, the total duration of the muscular contraction is longer than a normal ventricular beat. Furthermore it produces a larger deflection, because in a normal beat the impulse from the auricles reaches both ventricles simultaneously, and these synchronise and

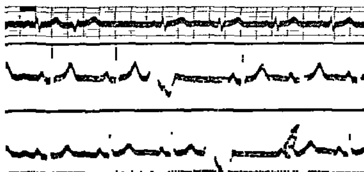


FIG. 13.—THE TRACING IS NORMAL EXCEPT FOR THE PRESENCE OF VENTRICULAR PREMATURE BEATS

The curve of the premature beat is characteristic, being in two leads out of three of much greater amplitude than that of the normal ventricular QRST complex. The premature beat curve is in these two leads composed of two parts, the first roughly corresponding to the QRS part of the normal ventricular curve, and the second part corresponding to the S-T portion. The first part of the abnormal curve is of briefer duration than is the second, but is of greater amplitude. The second or T wave part of the abnormal curve is in these two leads in the opposite direction from that of the first part of the curve. The interval between the two normal complexes separated by the premature ventricular beat is exactly equivalent to that of two normal heart cycles.

cancel each other electrically to some degree. In a premature beat both the synchronisation and the cancellation are lost, and the result is a large swinging complex (Fig. 13). The normal P wave which occurs between these two normal cycles interrupted by the premature beat, is submerged by the latter, and is not seen in this tracing.

In the case of the auricular premature beats these arise away from the sino-auricular node, and cause an action current which therefore has an abnormal auricular course. If the origin of the premature beat is near the auriculo-ventricular node, the auricular contraction will be reversed, so that the P wave may be inverted (Figs. 14 and 15). But the impulse still travels from the auricle to the auriculo-ventricular node, and from this point the mechanism is normal, so that the ventricular contraction will be normal in shape and size, in

nearly every case Exceptionally, the ventricular tissues may not have quite recovered their conductive power The ventricular complex will then be atypical in shape

Both auricular and ventricular premature beats can be taken, in most cases to have little or no clinical significance Apparently they are evidence of some local myocardial irritability, but never of failure Their local character is proved by the constancy of their form and

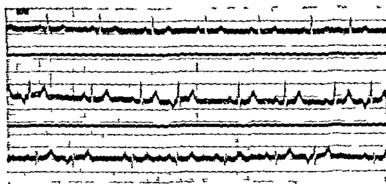


FIG 14 —AURICULAR PREMATURE BEATS SITUATED AT SOME DISTANCE FROM THE SINO AURICULAR NODE

The P wave is therefore inverted in Leads 2 and 3 because the contraction of the auricle produced by the premature stimulus is to some extent from below upwards The space occupied by the premature auricular beat and its subsequent QRST complex is less than that of two complete normal heart cycles At the end of Lead 1 an auricular premature beat is seen the P wave of which is nearly isoelectric and therefore nearly visible

therefore of their point of origin in any given case Sometimes two or three such foci may coexist and their number is then evidence of increased myocardial irritability To this extent they may suggest the imminence of fibrillation auricular or ventricular

Certain things influence the appearance, disappearance or frequency of premature beats Exercise by increasing the normal heart rate, and by making a normal beat premature to the irritable focus, usually silences the latter and removes the irregularity As the rate falls the premature beats return Posture may influence their appearance either by variation in the blood pressure or by variation in vagal or sympathetic tone Standing is apt to bring them out, and lying flat to repress them Gastric distension or irritation is also liable to produce them or to increase their number Some drugs are apt to evoke them—digitalis thyroxin and the social poisons caffeine nicotine and alcohol

Symptoms and Signs

All people have premature beats at some period of their life, but many never notice them. Sensitive individuals complain either of a bump or throb, or of a momentary sinking feeling, or of a pain. *The bump is caused by the strong beat which follows* the pause, shown in Fig. 12. Since the premature beat expels little blood, the normal beat which follows the pause has an accumulated quantity to

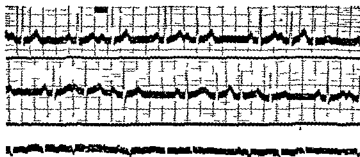


FIG. 15.—THE TRACING SHOWS FREQUENT AURICULAR PREMATURE BEATS

The ventricular or QRST parts of the tracing are normal in every respect. The premature auricular waves are not quite the shape of the normal auricular waves, this being particularly seen in Lead 3, where the normal P waves are visible, but those recorded prematurely are nearly isoelectric and barely visible. Since the premature P waves in Leads 1 [illegible]

exaggerated combined deflection of an abrupt rather unusual shape

contract upon, and expels nearly twice the normal amount. The blood pressure readings correspond, that of the premature beat being very low and that of the subsequent normal beat being much raised. The momentary sinking feeling, "like a rapid descent in a lift" in the words of one patient, is self-explanatory. The momentary "pain," if the history is sketchily taken, may be wrongly interpreted as anginal.

The chief sign is the pause at the wrist. During this pause auscultation over the heart can detect the sound of the premature beat, but this may be distant and audible only over some particular area, generally under or near the sternum. A weak premature pulse wave may correspond, but may be absent. In heart block, with dropped beats, there is a similar pause, but it is quite silent. Sino-

auricular block also gives a silent pause but the cardiogram shows no auricular and no ventricular beat in it

A premature beat may bring out pulsus alternans the alternating but otherwise normal beats following after immediately Pulsus alternans is a serious condition Where normal and premature beats alternate in a series the phenomenon is called pulsus bigeminus (Fig 16) Here the rhythm is irregular, in contrast to pulsus alternans Pulsus bigeminus has no especial significance

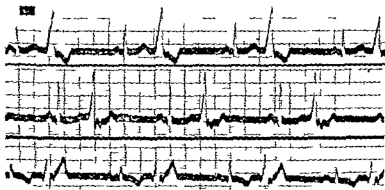


Fig 16

Each normal QRST complex is followed by *that of a premature ventricular beat* *This is sometimes known as pulsus bigeminus* It must be *distinguished from pulsus alternans* in which condition the second beat of the pair is not a premature or ectopic beat but is a beat which is perfectly normal electrocardiographically *In pulsus alternans all the* beats strong and weak are regular and *spaced at equal intervals from* one another

Premature beats *of no significance* are those that occur in the absence of symptoms of cardiac failure and without cardiac enlargement They may exist throughout a long life often in considerable number and will have no effect on longevity

Premature beats *of clinical significance* are those that occur in children with symptoms of acute or subacute rheumatism, and those of auricular origin that are found in mitral stenosis or in thyrotoxicosis where they may herald the onset of auricular fibrillation There are patients usually elderly in whom exercise increases the number of premature ventricular beats instead of abolishing them This group of premature beats is ventricular in origin, and is common in patients with myocardial degeneration During digitalis therapy premature ventricular beats are characteristic of overdosage, usually in the form of coupling

Of all types it may be stated without serious inaccuracy that when they occur with no other evidence of heart disease they can be ignored and that when such evidence exists it is of more importance than the premature beats

Treatment

Reassurance is the best treatment for premature beats This must follow a complete examination in which no other cardiac

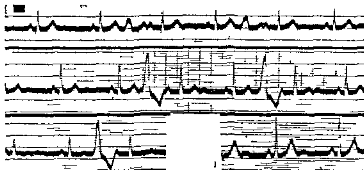


FIG 7—AN EXAMPLE OF A RARE CONDITION CALLED INTERPOLATED PREMATURE BEATS

The heart rate is slow 60 beats per minute and the premature beat occurs in between two normal cycles without preventing the development of the subsequent normal beat There is thus no compensatory pause Another interesting phenomenon is visible The rapid occurrence

abnormality can be discovered In addition it is wise to pay particular attention to any dyspepsia which may be present and to eliminate all sources of possible reflex gastric irritation In certain individuals certain articles of food may provoke the disorder Coffee strong tea alcohol spices twice cooked meat sausages are possible causes Over smoking must be controlled Drug treatment is useful Quinidine sulphate gr 3 t d s p c and atropine sulphate gr 1/200 to 1/100 t d s or in the form of belladonna can be given separately or simultaneously

Potassium bromide and potassium citrate not the sodium salt 15 grains and 30 grains respectively t d s p c in a mixture may be helpful Phenobarbitone as a sedative is valuable in nervously sensitive cases

CHAPTER XIII

PAROXYSMAL TACHYCARDIA

PAROXYSMAL tachycardia is very closely analogous in its mechanism and in some other respects to the premature beat The irregularity arises similarly from an ectopic focus either in the auricular or the ventricular or rarely the nodal tissues The electrocardiogram of one beat taken from paroxysmal tachycardia is indistinguishable from that of a single premature beat, whether auricular or ventricular



FIG 18

Indeed a possible definition of paroxysmal tachycardia is "a regular series of successive premature beats, occurring at a rate higher than that of the normal rhythm" Paroxysmal tachycardia is of the auricular variety in the vast majority of cases This is one of the commonest if not the commonest of all the cardiac irregularities The rare ventricular variety is noteworthy from the fact that it usually denotes serious myocardial disease

Fig 18, comparable with that illustrating the mechanism of premature beats, shows first three normal beats, then a short run of auricular paroxysmal tachycardia, then three normal beats then a short attack of ventricular paroxysmal tachycardia

Certain points shown in the figure need amplification The high rate in the paroxysm shortens the total diastolic period considerably, reducing the ventricular output during the paroxysm The auricular impulses, in auricular paroxysmal tachycardia, differ in origin from case to case, so that the corresponding abnormal P wave varies too, as with auricular premature beats The ventricular complex remains normal in shape The rate in the attack may be as low as 100, is most frequently around 120 to 160, and may be as high

as 190 The electrocardiogram (Fig 19) in the auricular variety shows a rapid series of regular normal ventricular complexes, the P waves, being smaller, are often submerged in these ventricular curves, but if the start of the paroxysm happens to be caught on the film the first P wave is clearly of a different shape from the normal, and may be distorted or flattened, or inverted, according to the position of the ectopic focus in the particular case

Isolated auricular premature beats are not uncommon in these

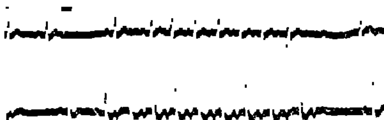


FIG 19—SHOWING TWO SHORT RUNS OF AURICULAR PAROXYSMAL TACHYCARDIA IN LEADS 1 AND 2

The complex of the normal beat shows an upright P wave in both leads In Lead 2 an abnormal focus placed well away from the sino auricular node caused a regular succession of rapid stimuli which produced corre

cases, and their P wave is similar to that found in the paroxysm when the latter is visible

The electrocardiogram of ventricular paroxysmal tachycardia consists of waves of wide amplitude, often diphasic, with no or little resemblance to the normal ventricular complexes

Ætiology of Auricular Paroxysmal Tachycardia

Auricular paroxysmal tachycardia is due to the activity of some focus in the auricular muscle, which is the origin of the ectopic rhythm A number of reasons combine to prove that this focus is an "irritable area' rather than the site of active disease

The condition is commonest in young adults in whom no other abnormality can be found It usually becomes less and disappears as years go by Between the attacks the individual is capable of normal activity

It is one of the commonest of the minor cardiac irregularities, yet

the attack is rarely observed by the physician, because the paroxysm is usually brief, lasting from a few seconds to a few hours as a rule, and the intervals between attacks measure days, weeks, or months Thus the probability of an attack occurring during the time of medical examination is remote A few patients suffer from more severe attacks lasting for several days Here the symptoms and signs become accentuated during the progress of the attack

The nature of the underlying lesion is uncertain In a few cases there is a previous history of acute rheumatism, scarlet fever, or diphtheria Mitral stenosis and thyrotoxicosis are occasional causes Attacks sometimes supervene during active rheumatic or diphtheritic carditis or in myocardial degeneration But certain factors are known to precipitate attacks Among these are gastro-intestinal disturbances or idiosyncrasies to strong tea, coffee, tobacco, alcohol, or fatty foods Vasomotor factors are also influential An attack can sometimes be precipitated by standing the patient up, after a period of recumbency, moreover it is easier to stop a paroxysm, by the measures described later in a patient who is lying flat

The significance of the auricular type is slight, and the disorder has little more than a temporary nuisance value The ventricular variety is more serious, both in young and in more elderly patients Death in an attack is extremely rare in the auricular type, but not uncommon in the ventricular

Symptoms and Signs

Since the actual paroxysm is not likely to occur during medical examination, and is therefore not directly observed, it is upon an exact history that the diagnosis will in most cases depend The usual story is that of an attack of palpitation with a noticeably abrupt onset This occurs at no especial time, and from no observable cause Suddenly the heart begins to race, throb, or flutter The palpitation may be felt in the left chest, near the apex beat, or at the root of the neck It may be described as a beating, a throbbing, or "like a fluttering" in the chest This persists for a varying length of time, and then as suddenly ceases But the cessation is not as exactly appreciated by some patients as the onset, for the effect on the heart is often, as it were, to stir it up, so that the rate of the normal rhythm immediately following the attack is considerably increased for a time The reason for the abruptness of start and finish is that the cardiac mechanism is suddenly switched from the normal guidance of the sino-auricular node to the abnormal mastery of an ectopic focus, placed elsewhere in the heart muscle At the

cessation, the regulation of the normal pace-maker is as suddenly restored

During the attack certain symptoms and signs develop The reduced output nearly always produces a sense of lassitude and some discomfort, from cerebral anæmia Fainting is rare, the heart rate being insufficiently raised Sometimes the association of a driven heart and a diminished left ventricular output combine to cause pain across the upper chest of the classical coronary or anginal type This may radiate in the typical manner Rarely, attacks last for a number of days, and produce the symptoms and signs of gross congestive heart failure These symptoms and signs rapidly subside when the normal rhythm is restored

If examination is made during an attack the following physical signs may be observed The patient may appear worried, and even distressed The heart rate is found to be increased to 140 for example There is often increased pulsation in the neck veins, and this is to be expected in cases where the abnormal pace maker is so placed as to cause the right auricle to contract from below upwards, or laterally This may also explain the feeling of abnormal neck pulsation

The apical thrust and the heart sounds both suggest an excited, overdriven heart The rapid thumping action is quite unlike the quiet running action of tachycardia due to fever or to any other such cause It is more like that of the heart excited by recent exercise But the great characteristic of the disorder is the constancy of the rate, regardless of the effect of physical activity Whether the patient has recently been walking, whether the patient lies down, or has been resting flat for ten minutes, still the heart rate remains at the same rapid figure or within four or five beats of this This is because the new focus is ectopic, or away from the normal sino-auricular node which is the point at which the vagus and sympathetic chiefly influence the rate of the normal heart In paroxysmal tachycardia this guidance is temporarily lost The blood pressure during the attack is lowered The sphygmomanometer may also reveal pulsus alternans, the difference between the alternate pairs of beats being about 10 to 15 mm of mercury In this condition pulsus alternans is devoid of the serious significance it has when the rate and mechanism are normal

Treatment

Treatment has two distinct aims to terminate the attack, and to prevent attacks arising The attack may be stopped either by physical

or by pharmacological means Firm pressure over the right internal carotid, in the neighbourhood of the carotid sinus, is sometimes effective Pressure over one or other eyeball may also succeed Also, the patient may learn to stop the paroxysm If a very deep breath is taken, and if then a forcible prolonged expiratory effort is made [illegible] is measure [illegible] He or she [illegible] and to use the same sort of pressure which is applied to overcome a constipated bowel motion but with the thorax quite full of air In some people an effervescing saline draught or one containing sodium bicarbonate, causes eructation and a simultaneous cessation of the tachycardia

Drugs are necessary only if this lasts for 12 hours or more Digoxin quinidine, and potassium salts are the most effective If digoxin is employed the best method is to give it in a single dose of 1½ milligrammes (six of the usual 0 25 mg tablets) Quinidine is best given in doses of 3 grains two hourly for ten doses Potassium salts can be given as pot cit 45 grains, together with pot brom 15 grains four hourly for three doses Other drugs worth trying are prostigmine 0 5 mg by intramuscular injection and mepacrine 0 4 gramme similarly given If the attack is still prolonged the treatment by these drugs must be more energetic but it is then not devoid of risk and is a matter for expert experience and responsibility

Acetyl β methylcholine (Mecholyl) intravenously is often efficacious 0 025 gramme in 10 c c of sterile water is given very slowly by one observer, while another watches the effect on the heart rate When this slows to normal the injection is stopped If too much acetyl β methylcholine is given a generalised convulsion may occur Intravenous quinine 10 grains in 10 c c saline, is also efficacious in some cases It must be given very slowly and discontinued as soon as the paroxysm ceases

Treatment directed to the avoidance of attacks includes attention to dyspepsia and to the thorough mastication of food, and drug administration But prophylactic drug treatment is not justifiable unless attacks occur sufficiently frequently or seriously as to warrant this Quinidine sulphate in doses of 3 grains or 5 grains t d s , p c , is the most efficacious drug Its action is sometimes improved by the simultaneous exhibition of atropine sulphate gr 1/200 to 1/100 t d s , p c , by mouth, or an equivalent amount of tincture of belladonna Digitalis in the usual standard maintenance dose is valuable in some cases Potassium salts, in doses of 45 grains t d s , p c , are sometimes worth a trial

VENTRICULAR PAROXYSMAL TACHYCARDIA

Ventricular paroxysmal tachycardia is an infinitely rarer but a

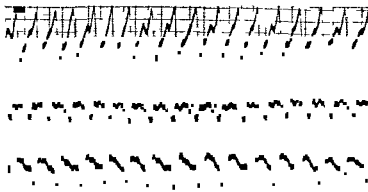


FIG 20

The tracing shows a series of complexes occurring at a speed of 200 beats per minute The splayed-out and simplified shape of the individual complex would suggest a ventricular premature beat if such a complex occurred alone The sudden succession of a series of such complexes occurring regularly suggests the presence of ventricular paroxysmal tachycardia The slight variation in the shape and in the regularity of the waves is not uncommon in ventricular paroxysmal tachycardia

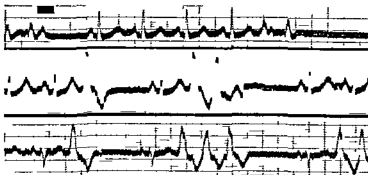


FIG 21

Normal P and QRS complexes are present in all leads Premature ventricular beats are also visible in all three leads occurring in runs of two or three Such runs of three constitute brief paroxysms of ventricular paroxysmal tachycardia The mechanical relationship between premature ventricular beats and ventricular paroxysmal tachycardia is apparent from this curve

much more serious condition, and is usually a symptom of coronary ischaemia and myocardial degeneration Death in an attack is not

uncommon In either type, when the length of the paroxysm induces congestive heart failure, the treatment of this is on the usual lines

The symptoms and signs of the ventricular type are usually indistinguishable from those of the auricular variety, but the patients are generally older In some of the ventricular cases the rhythm, during the attack is not strictly regular But the diagnosis can only be settled by the electrocardiogram (Figs 20 and 21)

The treatment is identical with that of the auricular type, with the following reservations During the attack intravenous acetylcholine preparations are best avoided Digoxin is safer and usually as efficacious as quinidine, but must often be given in large doses, such as 2 milligrammes daily, in divided doses, for several days

CHAPTER XIV

AURICULAR FLUTTER

Mechanism

THREE parts of the heart are involved in the mechanism of auricular flutter the auricles, the bundle of His, and the ventricles The auricles are the site of the abnormal rhythm whose effects are



FIG 22—A TYPICAL CURVE OF AURICULAR FLUTTER

[illegible]

unchanged from those occurring in the same patient before and after the attack of auricular flutter The T waves are buried

transmitted via the bundle to the ventricles, which are thus driven by the abnormal auricular stimuli

The focus from which the abnormal rhythm originates is in the neighbourhood of the large veins, usually one of the caval veins The impulse circulates round this rather as a spoon does around the bottom of a cook s mixing bowl The rate of circuit is usually from 200 to 300 per minute, and the path of circuit is constant in any given case As the excitation wave circulates it activates a wave of contraction in the auricular muscle This wave spreads out and through the muscle of both auricles, in much the same way as the stirring spoon flings up a wave of liquid batter against and around the walls of the bowl, but of course the auricular walls contract

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These contractions are real physical events measurable by a polygraph as forcible waves in the jugular pulse This is one point in which auricular flutter differs from auricular fibrillation, in auricular fibrillation the chambers have lost their power of coordinated and effective contraction and remain in a state of flickering distension Each wave reaches the upper end of the bundle of His down which it is transmitted, but only a proportion of the waves traverse the bombarded and fatigued bundle so as to reach the ventricular muscle

The bundle fails to transmit every impulse owing to the state of refractoriness or fatigue in itself and usually only one out of every



FIG 23 —AURICULAR FLUTTER LEAD 2 ONLY IN WHICH THE RESPONSE OF THE VENTRICLES IS NOT REGULAR

The second and third ventricular complexes respond to every other beat of the fluttering auricle The fourth responds to the fourth preceding auricular stimulus the fifth to the third and the sixth to the second The seventh responds to the fourth and the eighth to the fifth auricular stimulus There is in other words a very varying degree of heart block present and the rhythm of the ventricles and therefore of the radial pulse is very irregular

2, 3, or 4 auricular impulses reaches the ventricles (Figs 22 and 23) Thus a state of 2 1 or 3 1 or 4 1 block is present This constant proportion usually persists for long intervals in any given case, but in some patients the block varies so that, whereas the ratio of block may be for the most part 3 1, in occasional increase or decrease in the block occurs As a result the ventricle sometimes responds to the second and sometimes to the third or fourth auricular beat In this way the fundamental regularity of rhythm is broken momentarily here and there This momentary break in the otherwise regular rhythm is one of the clinical points suggesting the presence of auricular flutter, in a patient with an otherwise constant and non-varying tachycardia In an occasional case the rhythm of the ventricles may seem quite irregular (Fig 23) *In auricular flutter* digitalis has a marked action on the partially fatigued and therefore very sensitive bundle of His, so that by its use the block can be easily increased to 3 1 or 4 1, with a corresponding fall in the ventricular rate

A rare event in flutter occurs when for a brief period the ventricle responds to every auricular beat, thus beating regularly at, for

example, 250 per minute (Fig [illegible] encroaches seriously upon the [illegible] the cardiac output falls very [illegible] cerebral anæmia occurs, and [illegible] or faints Flutter is thus one of [illegible]

The ventricular contraction [illegible] the ventricular part of the elect[illegible]



FIG 24—A CASE OF A[illegible]
In the first part of Lead 1 and in the [illegible] responds to every auricular stimulus Th[illegible]

recorded in the same patient when th[illegible] stopped The regular rapid beat of [illegible] *tracing, as a regular undulation,* each p[illegible] ing to an auricular contraction The[illegible] and 3

Ætiology

Mitral stenosis, slow progressiv[illegible] coronary infarction, and thyrotoxicosis auricular flutter Bronchial carcinom[illegible] direct irritation of the auricles Mitr[illegible] ways, by the presence in the auricle rheumatic myocarditis, and by the [illegible] upon the auricular wall These cau[illegible] change in the auricular muscle as t[illegible] but in some cases flutter occurs instea[illegible]

Myocardial degeneration, secondary to arteriosclerosis, is the cause of most cases of chronic flutter, and in some such cases the degenerative lesion here may be almost confined to the auricle, while the ventricular muscle remains good for many years In one such case the auricular flutter would not respond to treatment, but digitalis kept the heart rate down to 60 or less The patient lived with auricular flutter present, for years, and died at the ripe age of eighty-two In other cases where the ventricular muscle is more seriously affected the duration of life is shorter Coronary infarction may cause auricular flutter, but the abnormal rhythm is usually here more transient Thyrotoxicosis, by its irritative effect upon the myocardium, may induce flutter in an occasional case, although fibrillation of the auricles is a far commoner complication

In a few cases auricular flutter is found with no other evidence of heart disease Mechanical irritation may induce attacks of auricular flutter It can follow local trauma such as that caused by a bullet wound, and be associated with secondary carcinoma infiltrating the chamber wall Occasionally, when quinidine is being used to convert auricular fibrillation to normal rhythm it causes auricular flutter instead

Symptoms

The symptoms depend upon whether the ectopic rhythm occurs in brief paroxysmal attacks, or whether it is of longer duration In the former type of attack the symptoms are indistinguishable from those of paroxysmal tachycardia There are intervals in which the patient remains in normal health, but periodically the flutter supervenes The duration is a matter of hours or days The onset is abrupt and so is the cessation The patient suddenly feels the heart begin its rapid beat, the sensation being usually felt over the præcordium, at the apex area, or deep in the root of the neck Simultaneously a feeling of general lassitude and incapacity occur Dyspnœa is present upon only slight exertion Pain over the chest may occur and may radiate to the arm The symptoms are due to the fact that the forced rapid beating of the ventricle gives less time for diastolic filling, with the result that the blood supply to the brain, to the systemic area generally, and to the coronary arteries is curtailed The supervention of 1 1 block may produce fainting in the manner previously described

When the auricular flutter remains for weeks, or months, the symptoms of palpitation subside soon after the onset and the patient settles down to a condition of chronic impairment of cardiac function

The history here is that the patient was in a normal state of health until a few weeks or months ago, but that since then lassitude, mental and physical, and undue dyspnœa on exertion have been noticed In many cases œdema of the feet and other signs of congestive failure begin to appear.

Signs

The signs of auricular flutter are a persistent and unchanging tachycardia, possibly a fullness and pulsation of the neck veins and, at a later stage, the manifestations of congestive failure The most characteristic sign is the constantly high ventricular rate It is noticed that the heart rate does not vary during the physical examination It remains, for example, at about 100 whether the patient is standing, sitting, or has been lying flat for ten minutes The rhythm usually remains regular, but occasionally this absolute regularity is broken momentarily, by a variation in the degree of block Rarely there is more irregularity, but exertion tends to make the rhythm more regular again, an opposite effect to that seen in auricular fibrillation Rarely, rapid regular pulsations at the auricular rate may be seen in the neck veins

Treatment

Attention should always be directed first to the possible exciting cause Obvious thyrotoxicosis or coronary infarction are easily diagnosed, but each of those two conditions may be more or less latent The treatment and the prognosis will be greatly influenced by the presence of either condition There are two methods of treating the irregularity as such digitalis or quinidine Digitalis therapy is the better and should therefore be tried first If it fails quinidine can be used Digitalis has a curious action in flutter If given in therapeutic doses it first transforms auricular flutter into auricular fibrillation If it is then withheld the fibrillation ceases and is replaced by the normal cardiac rhythm and mechanism If the dose is insufficient the fibrillation reverts to the state of flutter The duration of the state of fibrillation seems to be inversely proportional to the amount of digitalis given, and in a number of cases where really large doses have been given the normal rhythm has returned without any intervening stage of fibrillation having been observed During the first few days of treatment the total daily dose of digitalis may be as high as 90 minims of the tincture, 8 grains of digitalis folia, or 1 5 mg of digoxin After three or four days at this level the doses can be slowly reduced Careful watch must be

kept for signs of intolerance The drug is given three or four times a day Should this procedure fail, it should be given a second trial after an interval of one or two weeks If it is still ineffective quinidine sulphate should be tried The dosage and rules of administration are identical with those described in the treatment of auricular fibrillation If both these methods fail, digitalis should be given constantly to hold the ventricular rate at a resting figure of 60 or 70, for the rest of the patient's life, or for the duration of the flutter

Prognosis

In paroxysmal flutter the prognosis is excellent in those few cases where no other cardiac abnormality can be detected In the thyrotoxic cases also the outlook is good, for treatment of the underlying condition will cure the flutter But where mitral stenosis is the cause the probability is that the flutter will sooner or later be replaced by auricular fibrillation In coronary infarction the presence of flutter adds to the seriousness of the immediate outlook

In the commonest group, that due to myocardial degeneration, the outlook may be excellent in the absence of marked cardiac enlargement or of hypertension But when the heart is large, the blood pressure raised, or electrocardiographic changes indicating ventricular diseases are discovered, the prognosis is in proportion more grave Signs of congestive failure which does not respond to treatment, add much to the seriousness of the case Occasionally auricular flutter which has been present persistently, or in spells, for years ceases unexpectedly, and does not recur

CHAPTER XV

AURICULAR FIBRILLATION

A CIRCUS movement in the auricle provides the impulses responsible for auricular fibrillation The mechanism is similar to that of auricular flutter but differs from it in the following respects

In flutter the rate of auricular excitation is about 200 beats per minute, but in auricular fibrillation this is about 400 per minute In flutter the excitation wave regularly follows the same course, but in fibrillation the circuit is variable As the excitation wave is more rapid in fibrillation, the impulse reaches some myocardial areas before recovery has taken place Since the impulse must follow a muscle path which is not refractory, the position of the circuit in the auricle varies continually In auricular flutter the auricles contract and expel blood, in auricular fibrillation the auricles are distended and ineffective, although the excitation waves cause their surfaces to flicker

In fibrillation the many impulses originating in the auricle shower down upon the auriculo ventricular node and thus upon the bundle of His The majority of them are not transmitted by the conducting tissues, because these are refractory to most of such stimuli Not only is the number of impulses which reach the ventricle reduced, but their rhythm is quite irregular True regularity of the ventricular rhythm in auricular fibrillation is seen only in those rare cases where disease of the bundle of His has made this impermeable and produced complete heart block In all other cases there is an absolute irregularity both of ventricular rhythm and ventricular force When the heart rate is slowed, as by digitalis, the rhythm becomes less irregular, and sometimes the presence of irregularity cannot be demonstrated except by measurement of an instrumental tracing But any increase in the rate accentuates the irregularity The electrocardiogram (Fig 25) shows absence of the P waves of normal auricular systole Instead there are usually visible waves, in one or other lead, occurring as irregular tremors of the base line of the tracing Sometimes the position of the circus movement, in reference to the electrodes, is such that a regular series of F waves is seen, at a rate of about 400 per minute The ventricular

complexes show the same shape of QRS-T which was present in the patient before the onset of the irregularity

Auricular fibrillation may exist as a permanent abnormality for years or less commonly it may occur in brief attacks of paroxysms

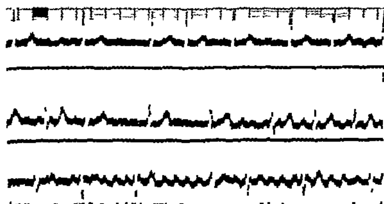


FIG 25 — AURICULAR FIBRILLATION

There are no regularly occurring normal P waves The ventricular

just before the sixth ventricular complex in Lead 2 In some of auricular fibrillation curves the fibrillary auricular waves are absent or small The fibrillary waves in Lead 3 bear a superficial resemblance to the P waves seen in auricular flutter but their rate is too high for flutter (420 per minute)

In the latter case it is identical as regards symptoms, and significance with auricular paroxysmal tachycardia

Causes of Auricular Fibrillation

The great majority of cases are caused by general disease of the auricular muscle A fibrillating auricle is incapable of any other form of activity It cannot propel the blood, and is in effect paralysed

The three commonest causes are mitral stenosis myocardial degeneration, and thyrotoxicosis Mitral stenosis causes the most untreatable variety of the irregularity, and thyrotoxicosis the most easily treatable Myocardial degeneration from coronary arterio-

sclerosis, may cause auricular fibrillation, as part of general severe cardiac disease, or it may produce it in a heart where the ventricular muscle is still good I have restored the normal auricular rhythm in such a patient by quinidine, and have seen the same patient, again with auricular fibrillation, after an intervening period of twelve years of reasonably good health with normal rhythm

Coronary infarction may be the cause of auricular fibrillation The fibrillation may be transient, and when it supervenes the pain of the coronary infarction may disappear

A less common cause of the irregularity is constrictive pericarditis (p 120) Rarely some local irritation of the auricular muscle may be the cause, examples are bacterial endocarditis with auricular mural vegetations, and secondary new growth from bronchial carcinoma

Drugs may produce the irregularity, digitalis and thyroxin, in large doses, are examples Occasionally a young healthy adult is found to have auricular fibrillation, with no other symptom or sign of heart disease Such cases need careful evaluation, but they may remain well for years

Symptoms

When auricular fibrillation begins a patient is immediately conscious of the irregular beating, running, and thumping of the heart, but after days or weeks these symptoms subside At the same time there is an immediate reduction in the cardiac output, for a fibrillating heart is mechanically less efficient than the same heart with normal rhythm Pain of a coronary type is practically never present with auricular fibrillation, and when this supervenes in the course of angina of effort the typical pain usually subsides The other symptoms present are those of the causative condition, or of heart failure

Signs

The fundamental sign of the irregularity is complete arrhythmia of the ventricular action This can be discovered by inspecting the neck vessels and the cardiac impulse, and by palpating the radial pulse Since, however, many of the smaller beats are too weak to lift the aortic valve cusps, auscultation over the heart is the only accurate means of detecting the rate per minute and the nature of the irregularity

The higher the heart rate the less likely are all the ventricular

beats to reach the wrist Thus the pulse deficiency is greater in failure

In auricular fibrillation the pulse rate figure is very misleading and the heart rate should be recorded by stethoscope instead of by the pulse rate, or in addition to it The sphygmomanometer demonstrates the great variation in the strength of those beats which reach the arteries So great is this variation that very accurate systolic and diastolic readings are impossible unless special methods of enumeration are undertaken But very fair average figures for the blood pressure can be obtained, so that the presence or the degree of hypertension can be recorded

The great clinical test of the presence of auricular fibrillation is exercise, or indeed any other measure which sends up the heart rate It is highly probable that an irregularity which becomes more irregular from exercise is auricular fibrillation, and that one that is lessened or removed by exercise is not auricular fibrillation

Treatment

The treatment of established fibrillation is either that of controlling the ventricular rate by digitalis or that of abolishing the disorder by quinidine

Digitalis has been used for the treatment of heart failure since the days of Withering, but the mechanism of its action has been clear only during the present century Digitalis proper is obtained from the purple foxglove, and digoxin from the white variety "digitalis lanata The most useful preparations are digitalis folia tablets made from the dried leaf, tincture of digitalis and digoxin, a crystal

of the tincture

Digitalis owes its reputation to its dramatic action in heart failure with auricular fibrillation In the absence of this disorder there is no doubt that its effect is slight, and some doubt whether it acts at all The usual pharmacological description of a prolongation of diastole by digitalis, with a stronger systole, cannot be demonstrated clinically In auricular fibrillation digitalis depresses the conductivity of the bundle of His, and thus reduces greatly the number of impulses from the fibrillating auricle which pass to the ventricle This latter chamber thus becomes much slowed, and achieves a better filling and a bigger stroke output The coronary and systemic circulations are equally benefited

The auricles fibrillate more easily under digitalis for it decreases *their refractory period* It is therefore clear that the drug never diminishes the fibrillation as such Whether the ventricle is stimulated to contract better is a much debated point On the whole the evidence suggests that this is so to a limited extent so that the exhibition of the drug in failure with a regular rhythm is a procedure which may or may not help Digitalis certainly acts in some way on the ventricle for the T wave of the *electrocardiogram becomes* inverted in all leads after a good course of the drug (Fig 26)

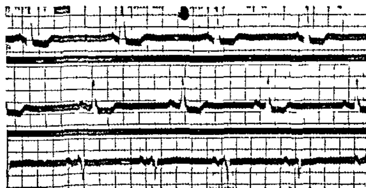


FIG 26 —THE EFFECT OF DIGITALIS IN NORMAL RHYTHM
The T wave is inverted in Leads 1 and 2 and flattened in Lead 3
Left axis deviation is also present

expensive The reasonable method is to give digoxin to produce digitalisation and digitalis folia to maintain it In a case of moderate severity digoxin is given by mouth in a dose of one milligramme, and this can be repeated in four hours and again after a further four hours if necessary *If the rate is already much less the subsequent* doses can be proportionately reduced In a case of severe failure with distress digoxin can be given intravenously, 0 75 milligramme in 10 c c sterile saline and this dose can be repeated in four hours

When the heart rate is lowered to 90 100 beats per minute digitalis folia can be given one grain three or four times daily by mouth This will reduce the apical rate further Eventually that dose of digitalis should be given which keeps the heart rate between 65 and

80 at rest This also is the result aimed at in ambulatory patients in whom half a grain twice daily may be a sufficient dose Each case must be assessed separately, for patients' requirements and toleration vary considerably

The tincture is used much less than it was for digitalis folia surpasses it in portability and digoxin in speed of action But there are cases in which it is still valuable One such was an adipose

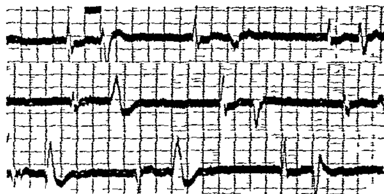


FIG 27—AURICULAR FIBRILLATION

Showing the effect of digitalis by the presence of coupled beats. The first beat of each pair has the normal origin but the second beat is a premature ventricular beat Although the normal beats are irregularly spaced the premature ventricular beat in every case follows its companion normal beat at a fixed interval of time throughout The premature ventricular beats in this case do not arise from the same focus as is obvious from their varying shape In other similar curves they may be identical in shape and origin

pregnant woman with persistent vomiting Her veins were hidden beneath fat and she would not retain a draught It was decided that she required 120 minims of the tincture The rectum was washed out and 240 minims twice the theoretical oral dose, was run in in two ounces of water This was repeated in four hours The effect on the tachycardia was rapid and good

"The proper dose of any drug is *enough*,' in the words of Drysdale *To use a drug fully and properly it must often be pushed to the limit* of tolerance This was well understood by Withering, whose writings describe fully the signs of digitalis overdose as we recognise them to-day They are malaise, nausea vomiting and coupling of the heart beat less often gastro intestinal disturbance, and rarely yellow vision Anuria sometimes occurs

Digitalis coupling is a condition in which a premature ventri-

cular beat follows every normal ventricular contraction (Fig 27) Thus there is a succession of beats in couples, the first of the pair being the stronger The interval between the normal and ectopic beat is constant Since the second beat of the pair is often weak it fails to register in the pulse An impression of bradycardia is then produced

Should the drug be further pushed this state of ventricular irritability may be increased, and may eventually lead to an attack of ventricular paroxysmal tachycardia, or even ventricular fibrillation and sudden death This is the reason why digitalis is said to slow the heart dangerously The mechanism is that of frequent premature beats, and not one of increasing heart block Heart block at the worst could only be complete, and this is compatible with normal activities Coupling and ventricular tachycardia due to digitalis can be treated by potassium citrate, 1 gramme four-hourly for three doses, or by quinidine sulphate, 3 grains four-hourly

The heart, like other organs such as the pancreas, the thyroid, and the kidney, is easily affected by infections such as influenza, bronchitis, or tonsillitis Its response to digitalis may be impaired by such infections If present in heart failure they must be actively treated

Quinidine Sulphate is used to restore the normal rhythm in suitable cases of auricular fibrillation It has a depressant action upon all forms of cardiac activity, but especially so upon the recovery of the muscle after contraction It lengthens the refractory period behind the circulating wave of auricular activity, and this refractory area of muscle eventually is met by the wave of excitation advancing upon it The situation is analogous to that of an old woman with a slowly lengthening skirt running round a lamp-post She is the excitation wave, and her train is the refractory period When she overtakes her skirt she falls down The cases where quinidine fails are cases in which the drug slows conduction unduly and has less action on the refractory period Here the speed of the old woman is reduced and she does not catch up with the skirt

Quinidine is given by mouth in an acid solution, and since it is rapidly excreted it is best given at intervals of two hours The drug is best made up as follows

Quinidine sulphate	gr 1
Acid hydrochlor dil	m 5
Aq chlorof ad	m 60
Unit dose m. 60	

On the first day a dose of 120 m. is given to test the patient's

sensitivity to cinchonism. If this is satisfactory the following programme is followed:

Day	Unit Doses	2 hourly for 8 Doses	Total Daily Dose of Quinidine
2	2		16 grains
3	3		24
4	4		32
5	5		40

It is safest to stop at a total dose of 32 grains per day, but where the patient can be kept under observation, and when the ventricular rate does not increase, the dose can be raised to 40 grains per day. It may be kept at 32 or 40 grains per day for 3 or 4 days. The rhythm often reverts to normal within 3–4 days of the start of treatment. Occasionally a second or third course is successful when the first and second have failed. The effect of quinidine seems to be better if the patient has been previously digitalised.

All patients receiving this dosage of quinidine should be nursed in bed, for the heart muscle is depressed and sudden death has occurred in ambulatory patients. Another danger is expulsion of clot from the freshly contracting auricle, with danger of cerebral, cardiac, or other types of embolism.

The choice of case is important. The best results are obtained in patients with auricular fibrillation who have no valvular defect, no gross failure, and no great enlargement of the heart. Myocardial degeneration and post operative cases of thyrotoxicosis offer the most favourable outlook. An occasional case of mitral stenosis may be suitable, but the irregularity here is prone to reappear within a few months. The most obvious contraindications are advanced mitral stenosis, much enlargement, and most cases of severe congestive failure.

Paroxysmal auricular fibrillation is sometimes seen in patients who are otherwise quite normal. Attacks occur at intervals, often of months or years, and between them no symptoms occur.

The symptoms are like those of auricular paroxysmal tachycardia, but on examination the rhythm is found to be completely irregular. The general treatment and the prophylactic treatment by quinidine is identical with that for paroxysmal tachycardia (p. 84).

Paroxysms of auricular fibrillation sometimes occur in individuals with mitral stenosis or with thyrotoxicosis as a preliminary to the onset of permanent auricular fibrillation. Runs of ventricular tachycardia occasionally occur in auricular fibrillation. The condition can be diagnosed only by the electrocardiograph. Quinidine in doses of three grains four times daily, is usually sufficient to control it. Digitalis treatment can be continued at the same time.

CHAPTER XVI

HEART BLOCK

WHEN the function of the conducting tissue of the bundle of His is impaired so as to delay or to obstruct the auricular impulse on its way to the ventricle, heart block exists The normal time needed for the transmission of this impulse varies between 0 12 and 0 2

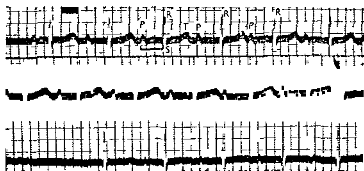


FIG 28 —HEART BLOCK STAGE OF PROLONGED P R INTERVAL WITHOUT DROPPED BEATS

The curve shows normal QRST waves but there is great increase in the length of conduction time from auricle to ventricle This reaches 0 31 seconds in this curve The patient was suffering from acute rheumatic carditis

second This time, which is known as the P R interval is lengthened by increases in vagal tone by drugs, or by disease Vagal prolongation of the P R interval is chiefly of academic interest The drugs producing heart block are digitalis and quinidine, neither of which in normal hearts can do more than prolong the P–R interval to perhaps 0 3 second Heart block due to congenital deficiency of the bundle occurs

In heart block the auricular contractions and the ventricular responses to them are normal in every respect, but delay in conduction, due to changes in the conductivity of the bundle of His, is present When there are no dropped beats the condition is called "latent heart block" since usually it cannot be diagnosed clinically This impairment of conduction may become greater, so that periodically

an impulse is obstructed completely, and fails to excite a corresponding ventricular contraction. This is the stage of "dropped" beats. The resting time which results from the pause in the ventricular rhythm allows the conducting tissues to recover to such an extent that the P–R interval following the pause may be normal. Subsequent impulses cause a progressive fatigue in these tissues so that the P–R time in many cases correspondingly increases till a beat is again

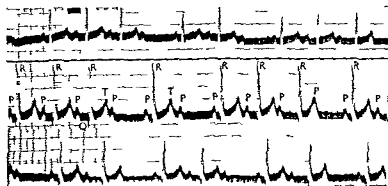


FIG. 29.—HEART BLOCK. STAGE OF DROPPED BEATS
In all three leads the P–R interval following each long pause is normal

transmit the impulse to the ventricle which therefore remains quiescent
From a case of acute rheumatic carditis

dropped (Fig. 29). Here occasional pauses occur in the ventricular action. These two types of heart block, latent block and that with occasional dropped beats, can be classed as low grade block.

When the bundle of His is more extensively damaged higher grades of block are seen in which alternate auricular impulses fail to traverse the bundle; these are labelled 2 : 1, 3 : 1, or 4 : 1 block. Finally no impulse at all can reach the ventricle, which then contracts at a rate of 30 to 40 beats per minute, the exciting stimulus arising from an independent focus, the idioventricular pace-maker, which is situated in the upper part of the bundle of His above its bifurcation.

higher, 40 to 60 beats per minute.

Low grade heart block usually results from an acute cause, such

as acute rheumatism or diphtheria in which the myocardium as a whole is poisoned Here the presence of heart block is a local indication of the diffuse myocarditis With recovery from the acute stage the heart block nearly always disappears Digitalis often *causes low grade block* *High grade heart block* on the other hand is usually a manifestation of a chronic local lesion involving the bundle



FIG 30—A CASE OF COMPLETE HEART BLOC

m
bea
cor

of His This may be arteriosclerosis of the artery to the bundle of His or a localised tertiary syphilitic lesion In these cases the rest of the myocardium may remain relatively unaffected particularly when arteriosclerosis is the cause

Symptoms

The symptoms of heart block are due either to the associated myocardial disease or to the presence of Stokes Adams attacks In the type associated with acute rheumatism or diphtheria the heart block itself is symptomless but its discovery adds to the gravity of the outlook since it is an indication of widespread and severe myocardial damage In a very few of these cases Stokes Adams attacks may occur

Where the block results *from a localised lesion the most striking*

Section V

PERICARDIAL DISEASE

CHAPTER XVIII

ACUTE PERICARDITIS

ACUTE pericarditis occurs in various ways In rheumatism it is part of the general carditis and in septicæmia it is secondary to the blood stream infection Septic pericarditis, caused by streptococci staphylococci and other pyogenic organisms, may occur by direct extension from a mediastinal focus, although it is remarkable that infection of the pleura rarely spreads to the pericardium Pneumococcal pericarditis may be a complication of a pneumonia or may be a primary acute disease, analogous to acute pneumococcal meningitis or peritonitis Uræmic pericarditis may be due to a terminal septicæmia or may be a silent non infective phenomenon symptomless and discovered in the course of examination The presence of pericarditis in coronary infarction is significant only as a sign of the infarct Tuberculous pericarditis may occur as rapidly, and with the same clinical prominence, as an acute tuberculous pleurisy with effusion or it may be latent and only reveal itself in the form of constrictive pericarditis after calcification has become widespread There is usually little fluid effusion in the acute types, but more in the pyogenic and tuberculous varieties

When the pericardium becomes acutely inflamed fever appears loss of lubrication develops and the outpouring of fibrin causes both friction between the visceral and parietal pericardial surfaces and stimulation of the sensory nerves Hyperæmia of these surfaces evokes a varying quantity of serous or purulent fluid A coexisting inflammation of the myocardium often develops

RHEUMATIC PERICARDITIS

This is a true manifestation of acute rheumatism as proved by microscopic demonstration of Aschoff nodes and other typical evidence of rheumatic inflammation It is the severest rheumatic manifestation for in it there is higher fever, a more rapid heart rate greater evidence of acute myocardial inflammation, a more rapidly

as acute rheumatism or diphtheria, in which the myocardium as a whole is poisoned Here the presence of heart block is a local indication of the diffuse myocarditis With recovery from the acute stage the heart block nearly always disappears Digitalis often causes low-grade block High-grade heart block on the other hand is usually a manifestation of a chronic local lesion involving the bundle



FIG 30 —A CASE OF COMPLETE HEART BLOCK

tracing also shows the changes typical of disease of the right branch of the bundle of His—namely, a deep S wave in Lead 1 and a tall R wave in Lead 3, both of these waves show widening and slurring

of His This may be arteriosclerosis of the artery to the bundle of His, or a localised tertiary syphilitic lesion In these cases the rest of the myocardium may remain relatively unaffected, particularly when arteriosclerosis is the cause

Symptoms

The symptoms of heart block are due either to the associated myocardial disease, or to the presence of Stokes Adams attacks In the type associated with acute rheumatism or diphtheria the heart block itself is symptomless, but its discovery adds to the gravity of the outlook, since it is an indication of widespread and severe myocardial damage In a very few of these cases Stokes-Adams attacks may occur

Where the block results from a localised lesion, the most striking

fact is that the presence of the associated bradycardia seems actually to spare the myocardium, and to assure it of a long diastole and therefore of a long period for good coronary filling between each beat Symptoms therefore may be unexpectedly slight and even absent

Stokes Adams attacks are due to complete ventricular standstill This is brought about by periodically diminished activity of the idioventricular node, the independent pace maker of the ventricles If the ventricles stand still for 3 or 4 seconds attacks of dizziness occur If the period of arrest is longer attacks of unconsciousness result, and if the duration exceeds 8–10 seconds generalised convulsions are seen The patient in the longer attacks becomes cyanosed, and the sphincters may be relaxed, death may result The attacks may recur at long intervals of weeks or months, at shorter intervals of days, or in rare cases many times in each day They seem to be more frequent at the onset of complete heart block As months or years go on they may become rarer, and disappear

Signs

In the first stage of heart block, where the transmission of the impulses from auricle to ventricle is only delayed and not inhibited no abnormal signs are present When beats are dropped there is a pause in the ventricular action during which the stethoscope can detect no sound over the heart This is in contradistinction to the pause in the pulse so often accompanying premature beats Here the sound of the premature beat is heard during the pause felt at the wrist In the rare irregularity known as sino-auricular block both auricles and ventricles periodically remain still and silent for a single heart cycle Sino auricular block is found in association with excessive vagal tone, and is of no clinical significance

Since the usual effect upon the heart rate of 2 1, 3 1, or complete heart block is to slow it, bradycardia is often the physical sign which draws attention to the existence of the condition Two other common causes of a slow regular pulse are alternate premature beats, and natural physiological bradycardia A premature beat may follow every normal beat, and may be of insufficient strength to raise the aortic cusps Here the pulse may be regular at for instance 40, but following every normal beat and pulse wave and in the pause, the premature beat can be heard through the stethoscope over the heart Normal physiological bradycardia is often found in athletes, but it may be seen in other types also

The bradycardia of heart block is either unaffected in rate by

moderate exercise, or tends to change abruptly Exercise may in some cases slow the rate by increasing the degree of block to one in which dropped beats occur *In other cases the increased sympathetic* tone may improve the impaired conduction of the bundle of His, and thus increase the rate The effect seems to depend upon how much of the block is due to physical damage and how much due to an increased vagal tone Normal bradycardia is easily affected by exercise, the rate increasing and the rhythm remaining regular

The other physical signs of complete heart block are characteristic With the patient lying flat the jugular veins tend to fill In them or at their point of issue from the thorax, in the hollow above the clavicle lateral to the insertion of the sternomastoid the regular pulsations of the normally beating auricle can often be counted, distinct from that of the slow ventricles, at a rate of about 80 beats per minute

Since the ventricular rhythm of complete heart block is independent, and since the ventricular rate has therefore no mathematical relation to that of the auricles, auricular and ventricular systoles will occasionally coincide When this happens the auricular venous pulsation is much exaggerated and at the same time the first heart sound at the apex is also abnormally loud In thin individuals the auricular systoles may be audible, usually at the apex in the periods of ventricular diastole

The blood-pressure readings in heart block with bradycardia do not reflect accurately the vascular condition present, and are deceptive If the heart rate is 30 beats per minute each systole is more forcible and more effective, the long diastole allowing very complete filling of the heart at each beat The systolic pressure is correspondingly raised A systolic reading of 180 or even 200 mm need not in such cases indicate a state of hypertension It is physiological and compensatory The diastolic pressure remains normal, at 80 or 90 millimetres of mercury

Treatment

Heart block requires no treatment as a rule Attention should be focused rather upon the causative disease Syphilis should be treated in the manner described on page 149 One point, however, requires mention Some patients with complete heart block suffer from congestive heart failure, with œdema There is no need to withhold digitalis in these cases, even though the regular rate is as low as thirty Digitalis is never dangerous because of its effect upon the bundle of His, for at the most it could only produce complete block, and here complete block already exists More than complete

heart block is an impossibility. The bradycardia of digitalis poisoning is caused not by heart block but by multiple ventricular premature beats, as in coupling, and is an expression of myocardial irritability, to push digitalis in such a situation is to risk producing ventricular fibrillation. In heart block with congestive failure, therefore, digitalis is of use by its direct action upon heart muscle, and it can be given in the usual doses.

Stokes-Adams attacks require treatment directed to restore the irritability of the idioventricular node. This is best achieved by sympathetic stimulation. Adrenaline by subcutaneous injection is the most effective drug. An injection once or twice a day of 0.25 to 1.0 c.c. of 1 in 1,000 watery solution will in many cases suffice to keep the patient free from attacks. The optimum dose can be found by experiment in each case. The writer has seen a patient thus treated over a period of four years who remained free from attacks so long as the single daily injection was given. Another patient with severe diphtheritic myocarditis developed heart block and suffered Stokes-Adams attacks every few minutes. Here heroic doses of adrenalin, 1 c.c. 1 in 1,000 solution at half-hourly intervals, controlled the attacks for the few days of his survival. Adrenalin in oil may be useful by reason of its prolonged action.

Ephedrine by mouth in some cases controls Stokes-Adams seizures. It should be given in doses of gr. ½ to gr. 1½ t.d.s., p.c. Barium chloride has been advocated for the same purpose. I have not personally observed any success in its use. Thyroid extract, as an augmentor of sympathetic tone, may occasionally be helpful. Doses of gr. ½ to gr. 1½ t.d.s., p.c. can be tried.

CONGENITAL HEART BLOCK

Cases of symptomless complete heart block are occasionally found in young individuals. This is generally of congenital origin. Many cases are associated with a congenital patency of the interventricular septum, but since the defect is anteriorly placed in the septum, and the bundle posteriorly placed in it, it is probably inaccurate to assume the two conditions are related anatomically. The ventricular rate is higher here than in the acquired types of heart block, figures of 40 beats per minute and more being usual. Such patients rarely have symptoms. Very occasionally Stokes-Adams attacks appear. Sudden death is a rare but definite possibility in congenital heart block. The treatment differs in no respect from that employed in acquired heart block.

CHAPTER XVII

BUNDLE BRANCH BLOCK

BUNDLE BRANCH block is due to the presence of a lesion in the septal myocardium so placed as to interfere partially or completely with the conducting power of either the left or right branch of the bundle of His In such a heart the auricular impulse travels at the normal speed through the uninjured branch and the corresponding ventricular muscle contracts normally The impulse down the injured branch is either delayed or obstructed so that the muscle normally served by it fails to contract at the proper time Eventually the passive muscle receives either the delayed impulse through the damaged branch or a direct impulse from the already contracting opposite ventricle The two chambers therefore contract asynchronously and as a result the ventricular contraction regarded as a [illegible]

[illegible]

myocarditis and is occasionally seen in acute rheumatism It may be produced by a coronary infarct In diphtheria the lesion is a very serious one and cases rarely recover The block may subside in acute coronary cases the QRS part of the cardiogram reverting to normal after some months In diphtheria and rheumatism a right bundle branch lesion is commoner but in coronary cases a left Chronic coronary disease is responsible for most of the cases of chronic bundle branch defect Syphilis is a rare cause A majority of the chronic cases show a lesion of the left branch

The clinical characteristics are as follows The lesion is commoner [illegible]
the bundle [illegible]
and after th[illegible]

There is one clinical sign suggestive of the condition This is due to the asynchrone of the two ventricles The hand placed firmly over the apex may feel a double thrust and the stethoscope may or may not pick up a reduplication of the first sound When this

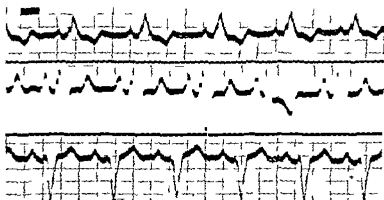


FIG 31 —LEFT BUNDLE BRANCH LESION

In this condition the impulse reaches the upper part of the bundle of

complex in Lead 2 is a premature ventricular beat

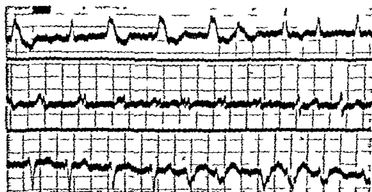


FIG 32

This is a rare but interesting curve showing temporary failure of the impulse to traverse the left branch of the bundle of His The first

reduplication is detected in a patient v (pertensio presumably suffering from myocardia r tion a h branch defect may be suspected The it hypert with a labouring ventricle, may also cau (ı ole thrust double first sound, but the cause is differen (p 15-)

The electrocardiogram (*Figs 31 and* ^2 ı *left bundle-* defect shows widening of the QRS part of the tracing to over a second, and often to nearly 0 2 of a secon l The general sh the curve is that of left axis deviation so that the R wave is in Lead 1 and the S wave deepest in Lead 3 The T wav usually in the opposite direction from that of the R and S being inverted in Lead 1 and upright in Lead 3 In ad notching or slurring is often prominent in the QRS waves

In right bundle-branch block there are two typical varie curve (Figs 33 and 34) The first is analogous in all respects t of left bundle branch block, but the general shape of the curve of right axis deviation so that S is deepest in Lead 1 and R tal Lead 3 The T waves again are in the direction opposite to t the R and S waves, being upright in Lead 1 and inverted in L The QRS prolongation and notching are present The s variety of right bundle-branch lesion shows widening chiefly t S waves of Lead 1 and Lead 4 The peak of the R and its fa of the normal sharp variety

Prognosis

The outlook in chronic bundle-branch block is uncertair cases *survive the first year many of them live for five or ten ye* more The prognosis is said to be better in right bundle br block than in the case of the left branch lesion If no conside enlargement of the heart is present, and no other symptoms signs of disease or of failure, the outlook is fairly good some living for ten years or so If much enlargement, anginal dyspnœa, or other evidence of myocardial defect, should coexis outlook is bad

Diagnosis

The only differential diagnosis is between bundle-branch d and the Wolff Parkinson-White syndrome, a rare condition in w one branch of the bundle receives its auricular impulse slightl advance of the normal time This is effected probably by of an accessory conducting path, a congenital abnormality

mechanical abnormality is demonstrated firstly by a shortening of the P–R interval, and secondly by a widening of the QRS complex

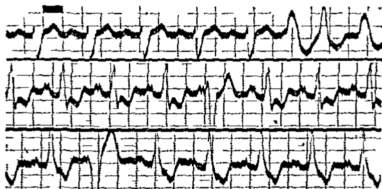


FIG 33.—RIGHT BUNDLE BRANCH BLOCK

There are two chief varieties of right bundle branch block. The first of these is practically a mirror image of that produced by left bundle

The present curve also shows premature ventricular beats three in succession at the end of Lead 1 and two isolated premature ventricular beats in Leads 2 and 3



FIG 34.—THE SECOND TYPE OF RIGHT BUNDLE BRANCH BLOCK

Also characterised by widened QRS complexes to more than 0.1 of a second. It also shows in Lead 1 a typical deep blunt S wave which is especially widened and slurred at its deepest point

Here the direction of the widening is in advance of the proper time, in bundle-branch defect it is delayed past the proper time. These

individuals in addition are subject to att paroxysmal tachycardia which is the usual cause of their medical advice The condition is harmless and the outlook (Fig 35)

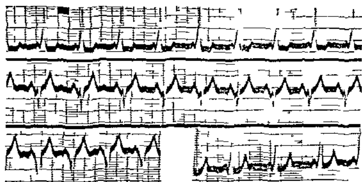
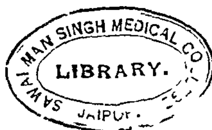


Fig 35 —Woolff Parkinson White Syndrome

The curve is typical showing the prolonged QRS complex in all leads and the much reduced P R interval which in this case measures 0 06 of a second When these cases suffer from their attacks of auricular paroxysmal tachycardia which they frequently do the electrocardiogram is typical of that condition



Section V

PERICARDIAL DISEASE

CHAPTER XVIII

ACUTE PERICARDITIS

ACUTE pericarditis occurs in various ways In rheumatism it is part of the general carditis, and in septicæmia it is secondary to the blood-stream infection Septic pericarditis, caused by streptococci staphylococci, and other pyogenic organisms, may occur by direct extension from a mediastinal focus although it is remarkable that infection of the pleura rarely spreads to the pericardium Pneumococcal pericarditis may be a complication of a pneumonia, or may be a primary acute disease, analogous to acute pneumococcal meningitis or peritonitis Uræmic pericarditis may be due to a terminal septicæmia or may be a silent non infective phenomenon, symptomless and discovered in the course of examination The presence of pericarditis in coronary infarction is significant only as a sign of the infarct Tuberculous pericarditis may occur as rapidly, and with the same clinical prominence, as an acute tuberculous pleurisy with effusion, or it may be latent and only reveal itself in the form of constrictive pericarditis after calcification has become widespread There is usually little fluid effusion in the acute types, but more in the pyogenic and tuberculous varieties

When the pericardium becomes acutely inflamed, fever appears, loss of lubrication develops, and the outpouring of fibrin causes both friction between the visceral and parietal pericardial surfaces and stimulation of the sensory nerves Hyperæmia of these surfaces evokes a varying quantity of serous or purulent fluid A coexisting inflammation of the myocardium often develops

RHEUMATIC PERICARDITIS

This is a true manifestation of acute rheumatism as proved by microscopic demonstration of Aschoff nodes and other typical evidence of rheumatic inflammation It is the severest rheumatic manifestation, for in it there is higher fever, a more rapid heart rate, greater evidence of acute myocardial inflammation, a more rapidly

developing anæmia, a higher leucocytosis and a greater immediate mortality It is usually a *manifestation which comes rather late in* the disease, and evidence of previous acute rheumatic attacks some years previously is generally available

The clinical *symptoms* are, great lassitude, distress and dyspnœa, and pain or discomfort either in the chest or referred to the upper abdomen

The *signs* are pallor and slight puffiness of the face and neck, with some orthopnœa, and the patient may present such a typical clinical picture that a facial diagnosis may often be hazarded, and later confirmed A very characteristic sign is a rather frequent, slightly irritative, dry and useless little cough, of which the patient appears to take little notice Nephritis and whooping-cough are the two other conditions which in children, cause a comparable pale puffy look, but in them the diagnosis can be easily made There is wide spread and acute inflammation of the mediastinum as shown post mortem by an œdematous or hæmorrhagic condition of the mediastinal lymphatic glands This might account for both the œdema of the head and neck and the cough

The veins in the neck may be rather full, and the arterial pulsation reveals the rapidity of the heart beat The cardiac impulse is generally seen to be displaced to the left, and is more or less visible according to the amount of fluid or fibrinous exudate present in the pericardium

On palpation the displacement of the apex beat is confirmed The thrills of mitral or aortic disease may be present, but these are often reduced in intensity by the flabbiness of the inflamed myocardium The friction of the two roughened pericardial surfaces *is often easily felt The other characteristics of this friction are* described later under auscultation

The percussion note shows an extension of the area of cardiac dullness to the left and to the right, which is due to the cardiac dilatation, probably also to the pericardial and mediastinal inflammation and to the intra pericardial fluid, although this latter rarely totals 300 or 400 c c in rheumatic cases The impairment of percussion note also extends upwards into the third and second spaces, to the left of the sternum

On auscultation the signs may be complicated by the presence of systolic or diastolic mitral or aortic murmurs, but in spite of this the friction sound is *generally unmistakable The sound may be grating* or shuffling loud or faint Its most valuable characteristic is the timing Whereas a systolic valvular murmur starts instantaneously

with the first heart sound, and a diastolic valvular murmur with the second, the systolic and diastolic pericardial friction sounds start slightly later than the heart sound in each case The sound may be twofold, in which case the double shuffle bears the same relation to the two heart sounds as the shuffle of dancers' feet does to the percussion sounds of a band, just a fraction late If the sound is triple, as it may be from the addition of a presystolic auricular friction sound the imagination can conjure up the waltz rather than the two-step The friction sound is usually heard better close to the left margin of the sternum than it is to the right, and is often loud under the manubrium

A further point to stress is that the intensity and the quality of the sounds are identical in volume and in character both in systole and in diastole A distant shuffle on one day may develop into the loud creaking rub in a day or two This persists for a period of days and then diminishes, often only leaving the slight shuffle which was heard at the onset If the patient's breath is held auscultation becomes easier

Pleurisy from whatever cause may give rise to "pleuro-pericardial" friction This differs from a true pericardial rub in that it usually coexists with a respiratory inflammation, and in that the friction although to and fro and related to the cardiac movements, is disposed along the anterior border of one or the other lung The cardiac murmurs of aortic-valve disease may simulate a friction rub at the base of the heart They are high-pitched, rushing and often distant Their instantaneous time relation to the first and second sounds and their permanence and unchanging character from day to day are characteristic

Certain pulmonary changes are common in rheumatic pericarditis They affect the lower lobes, most often the left The signs are those of consolidation impaired percussion note, and bronchial breathing which is often quite loud They have been ascribed to collapse of the lobe caused by pressure from a pericardial effusion It is more probable that they represent inflammation of a rheumatic nature, extending into the lung from the mediastinum, with more or less secondary collapse The chief reasons for this conclusion are that insufficient fluid is present in rheumatic pericarditis to cause such physical effects, that the changes start at the apex of the left lower lobe, and extend from there rather than from the base, and that some pathologists have demonstrated in the lung microscopical changes similar to those present in other acute rheumatic lesions Subcutaneous nodules are very common in

cases of acute rheumatic pericarditis Their presence should always suggest acute involvement of the mediastinal tissues

Heart block clinically obvious from frequent dropped beats is not uncommon in young adults attacked by acute rheumatic peri



FIG 36 —PERICARDIAL EFFUSION

Note the globular extension of the cardiac shadow in all directions from the level of the aortic knuckle downwards The shadow in its lower parts curves inwards near the level of the diaphragm Effusions of this degree are rare in rheumatic pericarditis they are common in tuberculous pericardial infection

carditis In spite of the severe nature of each of these complications complete recovery in such cases may occur

Cases of this disease are usually too ill for proper X ray examination but a portable film may suggest the presence of some fluid or fibrin by the more rounded and globular cardiac outline with corresponding reduction in the visible length of the aorta and the pulmonary conus

The electrocardiogram often shows a low voltage curve, with inversion of the T wave in Leads 1 and 2 S–T elevation reminiscent of coronary change may be seen If the bundle of His is affected a prolonged P R interval or dropped QRS–T complexes will occur

Treatment chiefly consists in rest in bed, in the position of greatest comfort the patient moving as little as possible Salicylates in the usual full dosage do no harm, whether they do good is un certain Oxygen is of great value, administered adequately by tent, mask, or double nasal catheter Distress and undue restlessness are greatly helped by morphia, even in children An ice-bag is sometimes applied to the præcordium The patient must in other respects undergo the usual long treatment of acute rheumatic carditis

The **prognosis** varies with age If a generalisation can be made, it may be said that at 26 years it is good even excellent, at 16 fair, and at 6 bad The presence of demonstratable mitral stenosis adds to the seriousness of the outlook

The **differential diagnosis** raises problems from the standpoint both of the physical signs and of the history

In addition to the friction sound, the cause of the cardiac enlargement has to be defined This might be due to dilatation In dilatation the apical thrust is weak also but percussion does not give the extension upwards so common in pericarditis The X ray is helpful (Fig 36) if the patient is not too ill for this In rheumatic pericarditis the shadow may not be typical of effusion, since this is often small in amount

In a child ill with active rheumatism the advent of high fever is very suggestive of pericarditis If pain in the chest is also present, pleurisy and lobar pneumonia must be excluded If pericardial pain *is referred to the abdomen, as it may be, it may lead the enthusiast* to a laparotomy and the removal of a normal appendix

Considerable difficulty often arises in distinguishing acute pericarditis from lobar pneumonia in children Fever, signs of consolidation over the lower lobe, especially the left, and a leucocytosis of perhaps 25 000 may be present in either condition Dyspnœa is about the same in each and sputum is absent in both But the onset of lobar pneumonia in a child is more sudden, the pain is usually definitely pleural, delirium is frequent and herpes labialis most suggestive Attention must be especially directed to the history of previous rheumatic manifestations, and of preceding sore throat or joint pains A mitral or aortic murmur will give a clue, but the presence of such a murmur does not exclude the possibility

of lobar pneumonia Finally, lobar pneumonia in a child rarely lasts eight days and usually terminates in four to six, so that prolongation of the situation beyond this should stimulate a review of the case The presence of rheumatic nodules is diagnostic The reaction to therapy may help Most cases of lobar pneumonia respond to sulphonamides, acute rheumatism does not

In the diagnosis of pericarditis the past history, as well as the present physical condition of the patient, is significant "Pneumonia" in the past history, may in reality have been acute pericarditis Such a patient can often remember that the attack was left sided that the cough was slight, and that the illness was prolonged This information may have a direct bearing on the present state

INFECTIVE PERICARDITIS

When pericarditis is part of a general septicæmia, the blood infection overshadows the picture, both in diagnosis and treatment Systemic chemotherapy or penicillin may sterilise the blood stream, and reduce the subsequent problem to that of treatment of a localised infection of the pericardium The situation then becomes one for co-operation between a physician and a surgeon with special knowledge and experience Paracentesis with penicillin instillation, repeated aspiration, or drainage, will be considered according to the degree and persistence of the infection, its loculation, and its course

TUBERCULOUS PERICARDITIS

This condition may arise in a patient who is known to have had pulmonary or pleural tuberculosis, or it may be a first manifestation The clinical characteristics are a rather insidious onset, with fever, increasing dyspnœa on exertion, and an irritative little cough There may be intrathoracic discomfort or pain

On examination the typical signs of pericarditis, already described, are noted, but friction, although present in the early stages, may have disappeared when the patient is examined The reason for this is that fluid is usually produced in greater amount than in pericarditis due to other causes This fluid produces other modifications in the picture the area of cardiac dullness is greater, both upward and laterally, a moderate effusion interferes with proper cardiac filling and causes distension of the neck veins, and a considerable one reduces the blood pressure, increases the heart rate, and lowers the pulse volume during inspiration, this inspiratory decrease being

called pulsus parodoxus The increased intra-pericardial pressure also evokes deep and increasingly rapid dyspnœa, which can be regarded not only as a respiratory response, but also as an effort to suck more blood into the heart from the systemic veins The physical explanation of this syndrome of increased intra-pericardial pressure sometimes called tamponade, is that at first the exudation of fluid into the pericardium causes no changes, but that when the increase begins to raise the intra-pericardial tension to a figure approximating to the venous pressure, auricular filling is interfered with This is one of the two indications for pericardial paracentesis The *other is to assist diagnosis by examination of the fluid in order to* determine whether it is serous or purulent, and what is its cytology and bacteriology By inspection alone, by looking at the pulse and respiration chart and at the patient s neck veins and respiration it is possible to determine whether paracentesis for the relief of pericardial distension is necessary These signs and symptoms of increase in the intra pericardial effusion are identical, whether the fluid is serous or purulent and whatever be its cause

Paracentesis of the pericardial cavity is not difficult Two measures require particular mention, and attention to them relieves the procedure of any feeling of strain attendant upon its rarity and upon the proximity of the needle to the heart Local anæsthesia must be complete, the skin, the subcutaneous tissues the neighbourhood of the ribs, the muscular and fascial tissues, and the pericardium itself receiving each its quota of novocaine, in methodical order and in adequate amount A period of four or five minutes must then be allowed for full anæsthesia to develop The apparatus must be so arranged that even if the needle impinges upon the heart *no harm can arise This is done by attaching the exploring needle* which should be of fairly wide bore (4 mm), to a length of rubber tube This in turn is fixed to a two way syringe If the needle is attached direct to the syringe and the latter is held in the hand the point becomes fixed and immobile, and constitutes a danger to the moving heart wall if the two should meet If the needle is free, except for its rubber-tube connection, it will readily move with the heart, if the two should touch, and will not scratch or wound it The whole system *must be very carefully sterilised, and filled with sterile* saline at the start On insertion of the needle the skin is pierced, then the soft tissues, and two resistances in turn are usually met, the pleura and the pericardium Since in pericarditis there may be an associated sterile serous pleural effusion, often small, the operator must take account of this and must persist until he is sure that the peri-

cardial cavity has been reached The best points of exploration are an inch or so inside the apex beat, an inch to the left of the sternum in the fourth space, and upwards through the diaphragm just below the xiphysternum The first two are the most usual

The **treatment** of acute tuberculous pericarditis is closely analogous to that of acute tuberculous pleurisy complete rest in bed, fresh air or open air, avoidance of direct sunlight high calorie and high fat diet with full vitamin content The progress must be watched by X-ray examination at regular intervals Improvement runs parallel with diminution both of fever and of the sedimentation rate, diminution in the pericardial shadow, and increase in weight Paracentesis is not indicated except for mechanical distress

The **prognosis** may be good with complete and permanent recovery but the future always holds the threat of a subsequent constrictive pericarditis

CHAPTER XIX

CHRONIC PERICARDITIS

THE results of chronic inflammation of the pericardium can be classified pathologically in four groups Silent ashesions may occur locally between the two pericardial layers in positions where they in no way embarrass the action of the heart The causes of these silent symptomless adhesions are unknown, mild attacks of rheumatic pericarditis may be responsible for some of them They are discovered during routine post mortem examination, the heart being normal in other respects Adhesion of the two layers may occur generally or locally in the pericardium in such a way as to constrict the heart, *constrictive pericarditis* Adhesions may form having the effect not of a constriction but of a local tethering of the heart to surrounding structures Adhesions may form in the pericardium as part of a general inflammation affecting one or more of the other serous cavities In *Pick's disease* peritoneal or pleural effusions may occur without the pericardium being involved

CONSTRICTIVE PERICARDITIS

Constrictive pericarditis in some cases follows a previous acute rheumatic pericarditis and in a larger number seems to be tuberculous in nature, but often there is no clue as to the origin The adhesions are fibrous at first, but after some years calcification may occur in them As the position of the adhesions themselves is often localised, and variable, so is that of the subsequent calcification The symptoms and signs depend upon whether the adhesions induce tension or traction upon the auricles or in the neighbourhood of the large veins as these enter the heart, or whether they embrace the heart, auricles, or ventricles in a gradually stronger contracting grip

Pressure upon the veins may partially obstruct the flow of blood from the superior or the inferior vena cava, and thus increase the local pressure in one or the other, whereas pericardial constriction interferes with proper diastole of the auricles or ventricles, or both, and so reduces ventricular output

Symptoms and Signs

The symptoms are so closely related to the anatomical effects of the disease that it is best to describe them together In the early stages, or when the lesion is localised to an ineffective position there may be no symptoms and the calcification in the pericardium may be revealed by a chest X ray taken *for some other reason* *Increasing* dyspnœa and lassitude, and a diminution in the amount of exercise that can be taken, are usually the first symptoms Simultaneously a fullness at the root of the neck increased by exertion may be felt Inspection of the neck at this stage may reveal abnormal distension of the veins An ambulatory patient who has walked to the consulting room may have visibly distended veins in the neck although no œdema of the feet or excessive dyspnœa are present This picture is never seen in congestive failure due to purely myocardial insufficiency, in which condition gross evidence of congestive failure appears in the dependent parts before the venous pressure in the jugular veins is increased to the stage of permanent distension It must be remembered, of course that tumours in the superior mediastinum may similarly obstruct the neck veins

In other cases of constrictive pericarditis the constriction interferes particularly with the return of blood from the abdominal cavity, and enlargement of the liver and ascites are the earliest and most prominent signs The return of blood from the systemic veins is cut down, but the return from the pulmonary vessels is less affected so that pulmonary congestion is avoided This is the reason why, in such circumstances gross physical evidence of congestive failure may be seen with little or no shortness of breath orthopnœa, or other distress

As the pericarditis becomes more chronic, inflammatory change begins to infiltrate the neighbouring heart muscle The thinner auricles are particularly susceptible to this and as a result auricular fibrillation is often produced It may be paroxysmal or permanent Attacks of paroxysmal fibrillation were the only symptom in a man whose hobby was mountaineering In his case the pericardial calcification was not yet constrictive, and was discovered during routine examination

Examination of the heart often reveals no clinical abnormality, but the organ is sometimes enlarged Diastolic shock may be felt over the præcordium, and accentuation of the second sound may be heard, although the blood pressure is normal Systolic murmurs may be audible and, *occasionally, a slight systolic creak usually at*

or outside the left border of the heart The apex beat may be impalpable a sign of more significance in youth than in age where

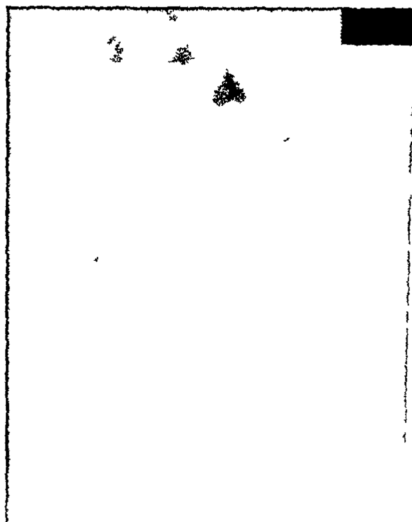


Fig 37 —Constrictive Pericarditis with Calcification
Left anterior oblique view

emphysema or a thick chest wall may normally be interposed between the hand and the heart The blood pressure is sometimes lowered a sign of lowered systemic output characteristic figures being 110 78 millimetres of mercury Pulsus paradoxus an inspiratory

decrease in pulse volume is sometimes measurable by finger or by sphygmomanometer

Radiographic and Cardiographic Examinations

Calcification of the pericardium may be seen best either in the antero-lateral or in the oblique views, and may occur at any point in the pericardium (Fig 37) It is most obvious when the calcified area is placed end-on to the film that is when it is visualised at the borders of the heart shadow in whatever plane this may be Calcification will be absent in the earlier fibrotic stages of the lesion, but diagnosis may still be possible from the other clinical and cardiographic evidence In addition the heart may be anchored to the sternum, not descending with the diaphragm on inspiration, and remaining constant in position whatever the position of the patient

The electrocardiogram (Fig 38) most typically shows flattening or inversion of the T waves in Leads 1 and 2 The curve is often of low voltage The Q wave may be prominent in Lead 3 Auricular fibrillation may be present

Treatment

Since the cause of the symptoms is mechanical, cure can only be by surgical removal of the structural defect Pericardectomy is the measure adopted Removal of the whole area of thickened pericardium is not often attempted the operation is usually restricted to such local removal as will free the venous openings into the auricles, or allow full auricular or ventricular diastole The surgeon and physician must cooperate fully in the diagnosis and treatment Each case presents a different problem Operation is most safely performed in children or in young adults As the disease becomes more chronic the inflammatory process invades the thinner auricles and right ventricle so as to make the definition of the walls of these chambers surgically difficult or impossible The stripping of the thickened pericardium from them is then an exceedingly hazardous matter Surgical treatment is also likely to be less satisfactory if cardiac enlargement or valvular disease is present Auricular fibrillation is a deterrent, if not a contraindication to operation Cirrhotic changes may be produced eventually in the chronically congested liver, and this also adds to the danger of surgical treatment Since the ætiology is so often tuberculous, it is clear that mechanical disturbance of an active caseous lesion is not without risk Tuberculous meningitis may follow this procedure

In subacute cases, too prolonged a wait may be impossible

because of the progressive congestive failure In such a situation the decision as to when to operate must be guided by the presence or absence of fever by the sedimentation rate and by the general symptomatic and physical state of the patient as well as by the cardiac picture

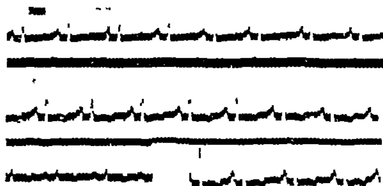


Fig 38 —A Typical Curve of Constrictive Pericarditis

In suitable cases the results are so successful that the possibility of surgical treatment should always be carefully considered A child or a young adult who has been confined to bed for months or for a year or more with ascites and œdema is able to return to a normally active and even athletic life after a few months

Diagnosis

Some patients with mitral stenosis may present a clinical picture very closely simulating constrictive pericarditis Dyspnœa some cyanosis increased venous pressure in the neck great liver congestion and ascites out of proportion to the œdema of the feet, all these signs may be seen in such cases But the murmur of mitral stenosis and the typical radiological findings are present Moreover gross cardiac enlargement is often seen in these atypical mitral cases The proved presence of mitral stenosis must be assumed to exclude that of constrictive pericarditis or at any rate to present a definite contraindication to any surgical treatment Otherwise a therapeutic disaster is highly probable

Cirrhosis of the liver may be suspected in other cases but the

youth of the patient, the absence of evidence of portal obstruction only, such as enlarged veins running from the level of the umbilicus up to that of the lower thoracic region, the absence of a sallow complexion, with venules on the cheeks, and the absence of morning vomiting and morning anorexia, are all points favouring the diagnosis of constrictive pericarditis

It must be remembered, however that chronic constrictive pericarditis, like very chronic mitral stenosis, may eventually produce progressive fibrosis in the liver, and often cirrhosis This may be the reason why ascites is present sometimes in these conditions when there is no œdema of the legs and feet The presence of cirrhosis of this type is one of the additional risks attendant upon surgical treatment of constrictive pericarditis It also makes such patients abnormally sensitive to drugs

POLYSEROSITIS

Polyserositis, or Pick s disease, may produce ascites or pleural effusion without any chronic pericarditis though this in some cases is added to the clinical picture In Pick s disease great enlargement of the liver and some enlargement of the spleen may occur from the deposition upon these organs of a thick coating of inflammatory fibrinous material Also there may be a fibrinous thickening or rolling up of the omentum When pericardial adhesions occur in Pick's disease the signs and symptoms may be the same as those found in the type of pericarditis with external adhesions described in the following section true constrictive pericarditis may also occur

ADHERENT PERICARDIUM

This term has been for long applied to cases in which adhesions are formed between the two layers of the pericardium, and through these between the heart and the surrounding structures, such adhesions being the result of previous acute pericarditis usually rheumatic *The signs and symptoms are said to result chiefly from* the formation of adhesions between the myocardial surface on the one hand and the sternum, ribs, diaphragm, or mediastinal structure upon the other These adhesions affect cardiac function most when they involve the ventricles If the ventricular muscle is bound *down for example, to the chest wall, it must add to its normal work* by pulling against the chest wall during systole Moreover, during diastole it is pulled rapidly out again to its resting position

Hypertrophy and dilatation are the result The largest and heaviest hearts are seen in these cases I have seen one which weighed more than the liver

Symptoms

So long as the hypertrophy is adequate the patient is able to live a normal rather careful life, but eventually dyspnœa and the other evidences of heart failure slight or severe, develop

Signs

In addition to the considerable hypertrophy and dilatation, signs appear which can be explained by the presence of the adhesion of the heart to the surrounding structures The apex beat, if carefully located and marked, is found not to shift when the patient rolls on to one side Around the apex beat the systolic traction of the heart pulls in the soft intercostal tissues, causing periapical systolic recession Adhesion of the heart to the diaphragm produces a systolic traction of this organ This movement is transmitted through it and its crura, and possibly through pleural adhesions also, to the soft intercostal spaces posteriorly, especially when the patient leans forward The recession is usually on the left side only, and it may be visible forward to the axilla It can occur on the right side In some cases the ribs also are drawn in by systole This is known as Broadbent's sign It should be remembered that when there is much hypertrophy a vigorous systole by itself may cause both the anterior and the posterior intercostal areas to show systolic recession in the absence of any pericardial adhesions

Inspiratory traction of adhesions upon the auricles may interfere with auricular filling, and produce inspiratory filling up of the neck veins, the opposite effect from that seen in normal individuals

The rapid pulling open of the ventricles in diastole, by the surrounding adhesions, may give rise to diastolic shock, in which the hand placed over the base can feel the exaggeratedly forcible closure of the semilunar valves An accentuated second sound accompanies this

The pulse may be affected when adhesions prevent proper auricular filling The pulsus paradoxus, an inspiratory decrease in pulse volume, being due to this cause

Radiographic and Cardiographic Examination

The radiograph shows great general enlargement of the heart, and possibly some exaggeration of the pulmonary conus if mitral

stenosis is present In addition, adhesion ' the heart to the diaphragm may cause a systolic jerking of th _an chiefly during inspiration, and adhesion to the sternum a l ig up of the heart or a failure to descend normally during inspi ation Systole and diastole may be poorly shown if the heart is very large The position of the heart may be fixed

The electrocardiogram is said in some cases not to show that variation in the size of the S and the T waves which occurs normally from change of position from the back to the sides This sign is not reliable though it can sometimes be demonstrated

Treatment

When it can be proved that adhesion is definitely present between the heart and the chest wall it is logical to treat this by removal of ribs over the area, so that the heart will have soft yielding tissues instead of unresisting ones against which to pull It is in a few cases successful in ameliorating symptoms

If cardiac failure is present it must be treated along conservative lines Digitalis should be administered particularly in the presence of auricular fibrillation, ascites is treated by paracentesis, and generalised œdema by ammonium chloride and mercurial diuretics

Section VI

VALVULAR DISEASE

CHAPTER XX

MITRAL DISEASE

MITRAL VALVULITIS is nearly always rheumatic in origin The acute or subacute rheumatic inflammation affects the shape, size, and flexibility of the mitral valve tissues in a variety of ways, but the result of these upon the efficiency of the valve is either a progressive narrowing of the orifice (*mitral stenosis*) or a distortion of the cusps, so as to prevent perfect closure without narrowing (*mitral regurgitation*) or a combination of these two effects, stenosis with regurgitation

The corresponding pathological changes are as follows Round the auricular margin of the valve the mural endocardium may become inflamed, at first acutely so as to cause swelling and œdema, and later chronically with the deposition of fibrous tissue and even of calcification, causing rigidity Secondly the edges of the two cusps may similarly become inflamed, stiffened, and distorted, so as to prevent their adequate closure during ventricular systole Thirdly the inflamed cusps' edges may become adherent to one another down their lateral margins This process spreads from the base of the cusps down along their adjacent free edges

The physical result of these processes varies according to the degree and type of the particular lesion Thus the chief effect upon the heart, and upon the circulating blood, may in some patients be predominantly due to a progressive diminution in the size of the mitral orifice, causing pulmonary congestion and a lessened output from the left ventricle In other patients the narrowing is only slight, although the valve components are chronically inflamed, fibrotic, and lacking in flexibility Here there will be seen only symptoms and signs due to the slight stenosis Finally, cases occur in which there is no actual narrowing at all, but in which valvular distortion has caused mitral regurgitation, with no pulmonary congestion and no effect on output This lesion is so relatively innocuous that it is only seen post-mortem when the patient has died

as a result of a superadded subacute bacterial endocarditis, or of some other fatal disease

Considering the lesion from the point of view of its effect upon cardiac work or the distribution of the blood it is clear that any degree of narrowing, however slight, must be labelled stenosis, and that any degree of failure of the rigid and deformed valve cusps to close must result in mitral regurgitation, however slight the leak Every case of mitral stenosis will have some degree of regurgitation, and many cases of clinical mitral regurgitation would reveal some slight narrowing of the valvular orifice if this could be examined and measured But, if the problem is judged by the physical effect the lesion may have either upon the distribution of blood in the circulation, or upon the consequent readjustment of the cardiac muscle, or upon the resulting symptoms and signs, there are two separate definable clinical entities stenosis and regurgitation

The argument of the purist that the one lesion never occurs without the other, although often valid in terms of morbid anatomy is not a practical clinical deduction

MITRAL STENOSIS

Mitral stenosis is produced by acute and subacute rheumatism, and by nothing else There are two common clinical varieties The first is that which occurs immediately following an obvious attack of rheumatic fever, acute rheumatism, chorea, scarlet fever, or tonsillitis with joint manifestations Here there is no doubt as to the relationship for the valvular lesion either develops within a few months of the causative rheumatic infection, or with a definite rheumatic past history The second clinical type is that which is found in adults, generally women between the ages of 30 and 45, in whom no definite history of acute rheumatism can be traced In these cases also rheumatism is the cause, occurring in recurrent attacks not severe enough to have been noticed A third, very much rarer type is found in patients in whom large vegetations form in the course of malignant endocarditis, the underlying abnormality in the mitral valve again being a scarring caused by rheumatism The recent vegetations are obstructive owing to their size The previous scarring caused no interference with blood flow

The changes in the valve cusps in classical mitral stenosis are as follows During the first acute inflammation the cusps become œdematous probably hyperæmic and somewhat stiffened From this stage perfect recovery is possible, but in many cases the process is

followed by a progressive fibrosis The inflammation often recurs, and the cusps slowly become thickened, shortened in both dimensions, and adherent at their adjacent margins This results in a buttonhole

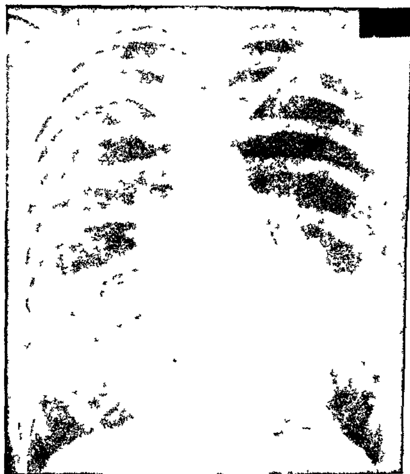


FIG 39 —MITRAL STENOSIS

Transverse cardiac diameter 13 5 cm Heart slightly enlarged Note the three prominences on the left border of the heart from above downwards they are the aortic knuckle the pulmonary conus and the appendix of the left auricle

deformity in adults and *one more of a funnel type in children* The changes here so briefly described may take years or even decades to develop

The effect on the circulation is to cause a progressive interference

with the flow of blood from the left auricle to the left ventricle This obstruction to normal blood flow produces changes both in the heart and in the pulmonary circulation It is simplest to follow these back from the mitral valve against the course of the circulating blood *The left auricle* is the first chamber to be affected and,

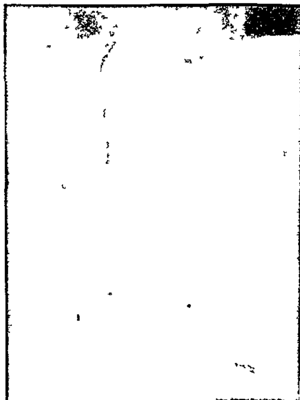


FIG 40—MITRAL STENOSIS SAME PATIENT AS FIG 39
Right anterior oblique After a barium swallow Note the posterior deviation of the œsophagus caused by left auricular enlargement

although some degree of hypertrophy occurs the outstanding permanent result is dilatation At first this may be impossible to diagnose, even radiologically, but soon it becomes visible on the X-ray screen Almost simultaneously there is a considerable increase in the amount of blood present in the pulmonary circulation, and this necessitates more activity on the part of the right side of the heart

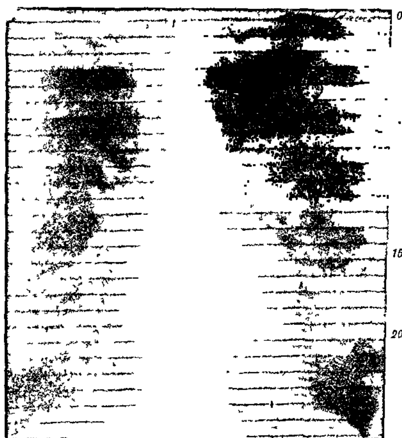


FIG 41 —KYMOGRAM ANTERIOR VIEW SAME PATIENT AS FIGS 39 AND 40

Note frame 7 aorta

frame 10 pulsation similar to that of aorta, and produced by pulmonary artery

frame 15, small pulsations of enlarged left auricular appendage

frame 20, left ventricular pulsations

A kymograph is an X-ray picture taken through a series of horizontal narrow slits These are stationary and the film slides down through a short distance Over the heart chambers systole causes a central recession and diastole an outward movement in the cardiac outline Over the great vessels these movements are reversed, systole causing an outward movement and diastole an inward recession

The first change in the left auricle is the increasing accumulation of blood in the chamber. This produces a correspondingly increased intra auricular pressure. Pressure within the auricle is

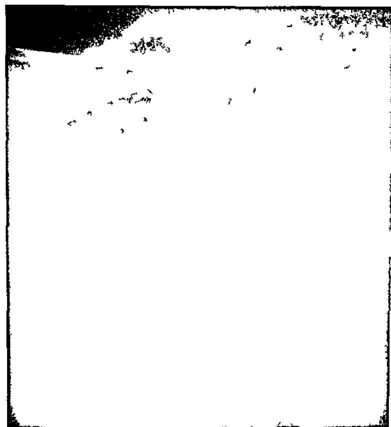


FIG 42 —MITRAL STENOSIS MIDDLE AGED FEMALE

Second radiological type. The heart is greatly enlarged. Compare with Fig 39 in which three prominences are seen on the left border. Note that the aortic arch is made almost invisible by the convex prominence of the left cardiac border below

raised most significantly during diastole when the heart as a whole is receiving its nutrient blood flow. The abnormal stretching of the left auricular wall gives rise to tension in the substance of the muscle of this chamber which interferes with its reception of coronary blood during diastole. The nutrition of the auricular muscle thus

becomes impaired slowly, both from the physical results of the progressive mitral stenosis and also possibly from recurrent rheumatic myocarditis The ultimate effect upon the auricles is to cause them to fibrillate, and to cease propelling blood After the onset of auricular fibrillation the auricular dilatation increases still more rapidly At first it involves chiefly the left auricle, but subsequently the right also In rare cases the left auricular enlargement may be so great that this chamber is visible radiologically to the right of the heart s outline, in the form of a spherical protrusion extending

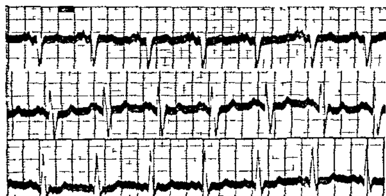


Fig 43 —Advanced Mitral Stenosis
The P waves in Leads 1 and 2 are notched and broadened Right axis deviation is present the S wave in Lead 1 being deep and the R wave in Lead 3 being tall These changes are caused by right ventricular hypertrophy

across from the left of the mediastinum Backward displacement of the middle and lower œsophagus, as visualised on the screen with the help of a barium swallow, is another constant radiological sign of left auricular enlargement and it occurs very early in mitral stenosis

Variations in the electrocardiogram run parallel with the changes in the heart muscle (Fig 43) The auricular hypertrophy shows itself by enlargement and notching of the P waves, while the rhythm remains normal Fibrillation of these chambers causes the P waves to disappear In their place fibrillary waves, constantly varying in size and in visibility, are seen between the ventricular complexes This variation is due to the constantly changing axis of the auricular circus movement The shape of the QRS–T complex of the ventricles remains identical with that present when the heart rhythm was normal

The pulmonary circulation can be considered next It is also the site of progressive abnormality The congestion which occurs early in the disease is associated with a decreased vital capacity, and the amount of congestion varies according to the general health of the patient with mitral stenosis, for example a severe cold will produce a temporary decrease in the vital capacity In the earlier stages the dyspnœa accompanying pulmonary congestion during exercise is the only clinical indication of any abnormality in the pulmonary circulation In the later stages the patient will suffer from a very considerable increase in shortness of breath, from orthopnœa, and, finally, from attacks of hæmoptysis, pulmonary œdema, and pulmonary infarction Cough caused by exertion is an occasional complaint Hæmoptysis is probably due to stasis at the lung bases, causing local thrombosis of the pulmonary vessels, and is not the result of embolism from the right side of the heart, as was once thought Early in mitral stenosis that part of the pulmonary artery nearest the right ventricle called the pulmonary conus, becomes enlarged This enlargement, together with that of the left auricle, causes a change in the radiological outline of the heart These enlargements fill out the left cardiac border so that the concavity between the aortic knuckle and the left ventricle normally seen in the antero-posterior view becomes filled in, and in advanced cases even become a convexity (Fig 42) A post-mortem indication of the increased pulmonary pressure is found in atheroma of the pulmonary artery *The right ventricle* becomes both hypertrophied and somewhat dilated in the later stages of the disease

The left ventricle, which lies, as regards the circulation, in advance of the lesion, is usually unaffected, and any change occurring in it is in the direction of atrophy rather than hypertrophy The reason for this is that the mitral stenosis prevents the left ventricle from ever being burdened with blood, and in the later stages of the disease actually relieves it of work

The chief circulatory results, therefore, of mitral stenosis are that the power of increasing the circulation rate during exercise is progressively diminished, and in the later stages the rate even during rest may be reduced below the normal The pulmonary congestion in mitral stenosis is much out of proportion to the degree of systemic failure

Signs

The physical signs of mitral stenosis vary with the severity of the disease In the early stages the patient may appear normal, later a

definite cyanosis with well coloured cheeks is common On inspection of the heart the cardiac impulse is frequently visible in the normal position If it is displaced to the left there is probably some additional lesion such as mitral regurgitation, aortic regurgitation, severe rheumatic myocarditis past or present, or an old pericarditis *accompanied by pericardial adhesions On palpation the position* of the apex beat is confirmed and the typical thrill or thrills of mitral stenosis can be felt It is helpful in describing the presystolic and the diastolic thrills and murmurs of mitral stenosis to think of the first of these by the label auriculo-systolic rather than presystolic and of the second by the label diastolic The presystolic thrill is caused by auricular systole and the diastolic thrill by the passive flow of blood from the left auricle to the left ventricle during *diastole*

The first thrill to develop is the presystolic or auriculo systolic, and to begin with, it is very brief and frequently difficult to determine As the stenosis becomes more pronounced this presystolic thrill becomes more definite and a diastolic thrill, which starts immediately following the second sound and the commencement of diastole, becomes palpable Since at the onset of auricular fibrillation the auricle ceases to be a propulsive chamber, the auriculo-systolic or *presystolic thrill disappears and the diastolic thrill only remains* The other characteristic on palpation over the apex is the slapping, forcible tap of the apex beat against the hand This is an early and a most valuable sign In the later stages the right ventricular hypertrophy can be detected from the heaving character of the cardiac impulse felt in the epigastrium The pulse is small in volume, and regular at first, and later, with the advent of auricular fibrillation, completely irregular

Examination of the heart by percussion confirms the position of the left and right borders and may also indicate the upward extension of the area of cardiac dullness, which results from the enlargement of the pulmonary conus and of the left auricle In such cases the note is definitely impaired in the third left interspace

On auscultation during the earliest acute stage of mitral inflammation, which corresponds to the inflammatory thickening and œdema of the mitral cusps, a reduplication of the apical second sound may be heard This sound is sometimes alternatively described as a short mid-diastolic murmur It is frequently not loud, and is not necessarily followed by the development of mitral stenosis It should be remembered that during the course of acute rheumatic carditis soft murmurs may come and go and therefore their evaluation

with regard to prognosis is difficult Six months or more may follow the acute inflammation of the mitral cusps before fibrosis begins In most cases this slowly progresses throughout the rest of the patient's life The total duration of this final stage is generally twenty or thirty years, and sometimes may be longer

The most typical murmur of mitral stenosis is the *presystolic* or, as it should more accurately be called, the *auriculo systolic murmur* This is due to the forcing of the blood through the mitral orifice by the contraction of the left auricle and it therefore immediately precedes the first sound or ventricular systole At first, when the lesion is slight, this murmur may be detected only after exercise or when the patient is lying down, and especially when rolled over on to the left side As the lesion progresses the murmur is present and audible in all positions, standing and lying, and it slowly becomes louder and more definite Palpation and auscultation should be applied separately, and with equal care to that phase of the heart cycle immediately preceding the first sound for in some cases a thrill is more easily detected than a murmur, whilst in others the reverse is the case

Sooner or later, according to the speed of development of the stenosis, the pressure in the left auricle during diastole becomes raised, causing an increase in the rate of blood flow from the left auricle to the left ventricle sufficient to produce *a thrill and a murmur during diastole* These are most apparent at the start of diastole when the rate of blood flow is fastest, and they diminish as the intra-auricular pressure falls and the intraventricular pressure rises When the heart rate is low they die away completely, leaving a silent interval at the end of diastole before the next first sound When the rate is higher the diastolic murmur is interrupted by the subsequent first sound It is in these cases that the inaccurate diagnosis of a presystolic or auriculo-systolic murmur in auricular fibrillation is made This is a contraindication in terms Such a condition is not possible the fibrillating auricle having ceased propelling blood The character of the diastolic murmur is that of a distant rumbling sound, like that of a wheelbarrow running over cobblestones some distance away When the auricles fibrillate this diastolic thrill and murmur may provide the only remaining clinical evidence of mitral stenosis *In some cases they are very hard to detect Both murmurs* of mitral stenosis, presystolic and diastolic, are strictly localised to the position of the apex beat, or just internal to it

Changes in the *first sound* are very important in the diagnosis of mitral stenosis These are an accentuation of the sound, the

development of a high-pitched quality of the sound, and some roughening of the sound Great attention should be focused upon the quality or the pitch of the first sound, for in some early cases this is almost the only means of differentiating between the first sound typical of *mitral stenosis and the accentuated first sound* so commonly heard in excitable young adults In the latter although the sound is accentuated and roughened, it retains its low pitch The sharp, high-pitched first sound is found quite early in the disease and remains throughout its course The association of the loud, high-pitched first sound with the brief presystolic murmur is very characteristic Experience is of great value in differentiating them from the sounds made by the normal but excited heart Lewis used to liken it to the bark of one's own dog, automatically but surely recognised

The *second sound* at the apex also undergoes a change, for it becomes progressively less and less audible, and in many cases disappears The explanation of this is uncertain Finally, the pulmonary second sound tends to become accentuated early and to remain in this state But in so many young adults accentuation of the pulmonary second sound is common this physical sign has, therefore less diagnostic value The characteristic change in the first sound and the murmurs of mitral stenosis are always more definite when the patient is lying than when standing, and more definite still when the patient is lying on his left side In doubtful cases auscultation in this position is always helpful for if the signs then become clearer mitral stenosis is probably present, and if the signs then become less obvious this lesion is probably absent Exercise, fever, or amyl nitrite also cause accentuation of these signs

Symptoms

For a long time during the early stages of the slowly advancing disease the symptoms of mitral stenosis are those of slight progressive heart failure, they are distinguished from other types of heart failure by the prominence of respiratory symptoms Dyspnœa occurs early Cough due to pulmonary congestion is frequently present, and may be accentuated by exercise In addition palpitation is often complained of, for the slapping character of the left ventricular action sooner or later draws the attention of the patient to his heart A result of this may be the development of the functional type of cardiac pain, which in some cases is acute enough to merit the label "*angina innocens*" Orthopnœa is almost invariable when the lesion becomes well advanced Rarely, the excessive

enlargement of the left auricle may cause hoarseness from pressure on the left recurrent laryngeal nerve and dysphagia from pressure on the œsophagus Hæmoptysis is common and in some cases appears to relieve the symptoms temporarily

Embolism is the cause of many of the most unpleasant complications of the disease It may occur while the heart rhythm is regular or with auricular fibrillation It is common in ambulatory patients *without congestive failure Clots form in the appendix of the* distended left auricle portions of these thrombi become detached and are carried as emboli into the systemic circulation In the brain such emboli may produce a hemiplegia which is often permanent In the kidney they may cause transient hæmaturia but renal and also splenic infarcts heal completely An embolus in the mesenteric vessels may give rise only to a transient attack of pain tympanites and the passage of blood in the motion after which complete recovery occurs but if the clot is large the result may be intestinal obstruction and trophic changes *in the intestine which necessitate surgical* resection The prognosis in such cases is bad

Congestive failure in this condition is as a rule similar to that seen in any type of heart disease In few cases ascites occurs to a degree out of proportion to the œdema elsewhere It may be early prominent and persistent and may precede the swelling of the legs and ankles The cause of this is obscure Possibly it is a result of an extreme degree of chronic liver congestion which has caused an unusual amount of hepatic fibrosis and even cirrhosis

The *degree of the mitral narrowing can best be evaluated* by attention to the following points The exercise tolerance of the patient as estimated by determining the amount of physical effort possible in the form of walking running stairs and games is most valuable As the stenosis increases it progressively deprives the left ventricle of the power of increasing its output of blood for these additional exertions Murmurs are misleading and occasionally may be typical and loud in an active athlete in whom the stenosis present must be so slight as barely to restrict the cardiac efficiency *Tachycardia even at rest is often present in advanced mitral stenosis* A resting heart rate of 60 beats per minute suggests that very little *narrowing is present The blood pressure is often lower rather than* higher than normal in advanced mitral stenosis and in the absence of aortic regurgitation the larger the pulse pressure *the less is the* stenosis likely to be The total absence of respiratory symptoms suggesting congestion indicates a slight rather than a more severe degree of stenosis

of the lesion, those of pulmonary congestion with hypertrophy of the right ventricle Inspection of the chest shows the apex beat to be thrusting in character, and in the late stages the impulse of the hypertrophied right ventricle can be felt in the epigastric notch A not unusual physical sign found by palpation is the presence of a systolic thrill at the apex When this accompanies a systolic apical murmur it is a definite indication of an organic lesion Percussion may confirm the enlargement of the heart to the left A systolic murmur which is loudest at the apex, and which is conducted into the axilla, is heard on auscultation This murmur is blowing, and in some cases has a musical quality It is also frequently audible at the pulmonary and aortic bases but is less loud there than at the apex It may also be heard behind, internal to the left scapula *This systolic murmur is a sign of mitral regurgitation, it is not a sign of mitral stenosis* The amount of regurgitation can be judged by the degree of left ventricular enlargement, and the chronicity of the lesion by the presence and the extent of right ventricular hypertrophy

The chief points in the **diagnosis** are as follows if there is no hypertrophy of the left ventricle, and no enlargement of this chamber, there is no mitral regurgitation present Certain other causes of systolic murmurs which can be heard at the apex have to be considered and excluded These murmurs can be due to organic, or to so called functional, causes The commonest organic lesions causing systolic murmurs at the apex are aortic stenosis, pulmonary stenosis and the presence of an interventricular septal defect In each case the position and the direction of conduction of the murmur are the important points upon which to concentrate In aortic stenosis the murmur becomes loudest as the aortic base is approached and in pulmonary stenosis the pulmonary base is the point of maximum intensity In defect of the interventricular septum the murmur is loudest to the left of the sternum in the third and fourth spaces Thrills frequently accompany these three lesions and are best felt at the point of maximum intensity of the murmurs Hæmic or functional systolic murmurs are common in young individuals (Chapter XXXV)

Enlargement of the pulmonary conus occurs much later in mitral regurgitation than in mitral stenosis, as seen on the X-ray film

Mitral regurgitation uncomplicated by stenosis is a lesion so innocuous that it is rarely seen in the post-mortem room unless the valve has been the site of a subacute bacterial endocarditis Probably in most cases it progresses to mitral stenosis, the increasing fibrosis gradually narrowing the valvular orifice For these two reasons

some authorities deny its existence, and m[illegible] ier the signs o[illegible] mitral stenosis and of mitral regurgitation [illegible] e fact that mitral regurgitation is rarely seen in the post mort[illegible] m merely means that it is relatively innocuous, not that it do[illegible] t exist Chickenpox also exists, but is not seen in the post mort[illegible]m room

CHAPTER XXI

AORTIC REGURGITATION

THERE are two main varieties of aortic regurgitation, the first follows distortion of the aortic valve cusps by disease, and the second results from stretching of the aortic ring by aortitis usually syphilitic Either *rheumatism or syphilis is the underlying disease in about* 90 per cent of all cases and atheromatous degeneration in about 10 per cent The relative incidence of rheumatism and syphilis varies greatly in different climates In Great Britain rheumatism is a commoner cause of aortic valvulitis than is syphilis There is also a small number of cases in which the aortic valve cusps are congenitally deficient the commonest defect being a bicuspid aortic valve *in these cases an infection of the abnormal valve cusps may* cause aortic regurgitation in a previously competent valve for the congenitally bicuspid aortic valve is functionally perfect and gives no clinical signs Aortic regurgitation influences the circulation in three ways the first is its effect upon the left ventricular muscle, the second its influence on the circulatory efficiency of the heart and aorta and the third that on the coronary flow

The left ventricle in aortic regurgitation has to perform additional work and has to accommodate additional blood This extra blood is directly proportional to the size of the valvular leak Since in most cases the amount of regurgitated blood is considerable and the distending force of the regurgitated blood upon the lax ventricle also considerable there is no valvular lesion which produces a greater degree of dilatation associated with hypertrophy of the left ventricle The amount of hypertrophy is usually proportional to the chronicity of the lesion but the dilatation may be in excess of the amount of hypertrophy in cases where the valvular leak occurs quickly, as in syphilis

In more chronic cases, generally rheumatic, the cardiac impulse is heaving and vigorous, and tends to be displaced downwards as well as towards the axilla In more acute cases, where a considerable leak has occurred rapidly, there has been no time for adequate hypertrophy, and the predominance of dilatation is shown by displacement of the apex towards the axilla rather than downwards,

moreover in the latter case the force of the apex impulse is poor and its position ill defined

The effect of aortic regurgitation on the *peripheral circulatory efficiency* is interesting It is best demonstrated by reference to a normal pulse tracing in which there is a good dicrotic notch That part of the normal pulse wave from the dicrotic notch onwards indicates the force of the elastic recoil of the aorta upon the contained blood, this recoil starting at the end of ventricular systole When this aortic recoil is lost, because the valve no longer holds up the diastolic pressure, the propulsive action of the aorta upon the blood during the latter part of the pulse tracing is also lost, so that, if the efficiency of the circulation is to be maintained, the left ventricle has to condense into a shorter period of time the whole of the work which would have been spread out over the full length of the normal pulse wave It is as though the left ventricle was throwing the blood forcibly at the systemic circulation on the chance that most of it would travel onwards, instead of being able to lever the blood up into the aortic chamber where the elastic recoil would carry on the work

It is evident that the degree of regurgitation in any given case is best estimated by the decrease in the diastolic blood pressure In a few cases, although the murmur of aortic regurgitation is present, the diastolic pressure is very little lowered This is particularly true in some cases caused by rheumatism Here the murmur indicates [illegible]

cases of aortic regurgitation is partly due to dilatation of the peripheral arteries and arterioles An exaggerated pulsation is seen in the carotids, brachials, and the other superficially placed arteries The capillaries are more open, so as to receive the periodic waves of blood thrown at them by ventricular systole But the opening up of these peripheral channels lowers the diastolic blood still further Capillary pulsation is the visible sign of this peripheral vascular dilatation

Aortic regurgitation has an effect upon the *coronary flow*, which is dependent upon the pressure present in the aorta during diastole, and is proportional to that pressure It is obvious that any rapid diminution in the aortic diastolic pressure will interfere with the coronary flow, and thus with cardiac nutrition

Aortic regurgitation is clearly a serious valvular lesion, for the left ventricle is less efficient mechanically and may be undernourished

K

The factor of myocardial disease is often added to the mechanical handicap

The **physical signs** of aortic regurgitation are as follows

On inspection the most constant finding is excessive pulsation in the arteries This is seen in the carotid vessels and may be sufficient to cause pulsation of the lobe of the ear, and is also obvious in the arteries of the arms and legs Ophthalmoscopic examination reveals pulsation in the retinal arteries If pressure is carefully applied to the nails, or over the lips by means of a glass slide, capillary pulsation can be demonstrated by a waxing and waning of the pink colour The apex beat is seen to be displaced downwards and outwards, and is visible over a much increased area *On palpation* the apex beat generally has a typical forcible heaving character The pulse shows a rapid rise and a rapid fall, and if the wrist as a whole is grasped and held upwards the typical "water-hammer" feel is detected

The murmur of aortic regurgitation is a rushing, high-pitched, diastolic murmur commencing immediately after the cessation of systole In syphilitic cases it is heard loudest in the second right intercostal space, and in rheumatic cases to the left of the sternum In both cases it is conducted down the sternum, most frequently along the left border, and is audible also out to the apex In rheumatic cases it may be very distant and difficult to detect, and may also be heard at the pulmonary base In a few cases, where the degree of regurgitation is considerable, a rather poorly defined presystolic or auriculo systolic murmur is audible at the apex beat or just internal to it This is the Austin Flint murmur, which is thought to be due to some semi-closure of the mitral valve during diastole The theory is that the regurgitating blood from the aorta moves the aortic cusp of the mitral valve towards its fellow, so that when the auricle contracts it does so through a half-shut valve Two other murmurs are not uncommon, that of mitral regurgitation and a systolic murmur at the aortic base The mitral regurgitation, when not rheumatic, is due to the great enlargement of the left ventricle which stretches the mitral ring The systolic aortic murmur is caused by the encroachment of portions of the diseased aortic cusps into the systolic blood stream But there is no adhesion of the aortic cusp edges and so aortic stenosis is absent Where aortic stenosis and regurgitation coexist the physical signs, cardiac and vascular, are predominantly those of aortic stenosis

may

size of the leak The blood pressure in the [illegible]

sometimes greater than that in the brachial arteries Typical brachial figures for a severe case might be 160 30

The **symptoms** of aortic regurgitation are frequently remarkable by their absence It is extraordinary how a syphilitic patient with an advanced lesion will complain of slight shortness of breath only for the previous few weeks when the extent of his lesion would indicate that the disease must have been present for some months The

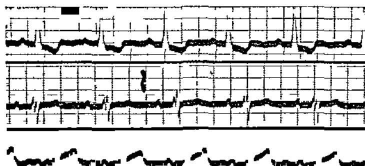


FIG 44 —CURVE SHOWING LEFT VENTRICULAR STRESS
This is from a well known jockey who had aortic regurgitation for

usual symptoms of early heart failure are generally present but in addition the patient may complain of the throbbing which is caused by the excessive vascular pulsation in the chest the neck or the arms Just occasionally it shakes the bed to such an extent that the patient s rest, or that of his wife, is disturbed

Pain of the angina of effort type is often caused in the syphilitic cases and more rarely in the rheumatic ones by the interference with the coronary flow It may be of the spasmodic angina type, and so severe as to demand surgical treatment, which is sometimes very successful in this type of case

When failure does begin it is often rapid and œdema of the feet with other signs of congestion indicates a poor prognosis Syncope is not uncommon in aortic regurgitation or aortic stenosis, and may be due to some abnormality of the carotid sinus mechanism resulting from the changed condition of the aortic pressure (Fig 44)

Treatment of aortic regurgitation is directed to the cause and to heart failure if present

The **prognosis** depends upon the cause Many rheumatic cases with slight aortic regurgitation may remain free from symptoms for years The arteriosclerotic cases generally manifest aortic regurgitation only when the heart and vascular system is grossly diseased in other ways Here too the valvular lesion as such is often slight and quite secondary In syphilis the outlook is grave unless *diagnosis can be made early in which case there is reasonable* prospect of arresting the disease In such cases a long period of good health can be subsequently enjoyed Other points to be taken account of in the prognosis are the extent of the valvular lesion as measured by the position of the apex beat and the diastolic blood pressure and the presence or absence of symptoms of heart failure and their degree

SYPHILITIC AORTIC REGURGITATION

When the aorta has become inflamed as a result of spirochetal infection the elastic tissue becomes in the course of time weakened and destroyed and eventually fibrous tissue replaces it The aorta therefore slowly becomes enlarged for stretched fibrous tissue is not perfectly retractile That part of the aorta which lies nearest to the aortic valve cusps is most affected The changes are less marked as the course of the vessel is followed through the thorax into the abdominal cavity Syphilitic disease of the abdominal aorta is rare

Syphilis distorts the aortic valve in two ways but in each case the basic lesion is syphilitic mesaortitis Aortic regurgitation may be caused either by simple stretching of the ring or by destruction of the valve cusps by the spirochete In either case the peripheral circulatory signs on the whole are indistinguishable from those produced by any type of severe aortic incompetence There are however a few clinical points worthy of emphasis in syphilitic cases In aortic regurgitation due to rheumatism the diastolic murmur is best heard down the left border of the sternum When the leak is due to syphilis the murmur is often louder at the aortic than at the pulmonary *base and in the second and third right interspaces* A possible explanation is that in rheumatism mitral disease is often present and this brings the pulmonary conus nearer to the left upper chest wall thus providing a solid conductor of sound to that area whereas in syphilis the elongated ascending aorta is pushed forwards nearer to the right upper chest wall

In some syphilitic cases the aortic cusps remain free from disease and flexible, and at the beginning of diastole they are filled sharply by the increased pressure in the aorta A loud second aortic sound then precedes the diastolic murmur Therefore, when aortic regurgitation is found to be present with a clear aortic second sound, the leak is probably due to stretching of the ring the valve cusps being normal A diastolic aortic thrill has also been noticed in this condition, when the normal state of the valve cusps and the dilated aortic ring were confirmed post-mortem

The second way in which syphilis produces aortic regurgitation is by extension of the syphilitic inflammatory disease of the aorta down the valve commissures into the substance of the valve cusps Here the cusps are thickened stiff, shrunken, and sometimes more or less destroyed Two further changes may occur secondary calcification resulting from an associated atheroma, and more rarely rupture of a weakened cusp The latter condition can be suspected where physical signs or symptoms, such as shortness of breath or pain, arise abruptly in an individual with aortic regurgitation A ruptured valve cusp in such circumstances frequently produces an exceedingly loud, roughened, bleating, or twanging murmur which may be noticed by the patient, or even by his wife, as he lies in bed

Another lesion present in syphilitic aortitis with regurgitation is the narrowing of the openings of the coronary arteries as they pierce the inflamed and œdematous or fibrotic aorta This narrowing results in diminution in the blood flow to the left ventricle and since in aortic regurgitation the enlarged ventricle needs more blood the diminution in blood flow is particularly dangerous The dilatation of the left ventricle also lengthens and enlarges the peripheral coronary vessels, and this is another factor which accentuates the handicap to the coronary circulation The rate of onset of the aortitis and valvulitis is often rapid and, as there is not sufficient time for hypertrophy, considerable dilatation ensues Therefore, the heaving apical impulse may be strikingly absent although the amount of the leak, as measured by the sphygmomanometer is great In such a case the symptoms of heart failure come on rapidly But in other cases although the leak is considerable the coronary flow remains good Here the striking paradox is the association of an advanced aortic leak with little or no symptoms of failure

The **treatment** of syphilitic aortic regurgitation is very successful, if the diagnosis can be made sufficiently early, but it is associated with certain risks If potent drugs such as bismuth, neosalvarsan or penicillin, are administered to such patients without previous

preparation the spirochætes may be destroyed locally around the coronary mouths to an extent sufficient to produce a considerable increase in the amount of local inflammatory reaction and œdema This may cause a severe reaction or even sudden death within a few hours of the first injection of the drug The danger can be much reduced if the patient is first treated for at least six weeks with potassium iodide and mercury by mouth The potassium iodide should be administered in doses of 10 grains every four hours with liquor hydrarg perchlor 30 40 minims and the dose of potassium iodide may be increased to 20 or 30 grains every four hours as soon as it is found that the patient has no signs of iodism and no dyspnœa or coronary pain while resting in bed At the end of this a course of twelve injections of bismuth injectio bismuthi B P should be given at weekly intervals and at the end of the bismuth medication intravenous neosalvarsan should be administered starting with a dose of 0 15 gram increasing to 0 3 and 0 45 gram at intervals of five days to a week The dose of 0 45 gram should not be exceeded The total course of neosalvarsan to be about 4 grams The Wassermann reaction should be then done This programme should be repeated after an interval of three months and similar courses should be given until the Wassermann reaction becomes negative Penicillin treatment is likely to prove valuable in syphilitic aortitis Large initial dosage should be avoided in order not to induce a reaction round the coronary mouths The dosage is a question needing expert decision

The dangers of not treating the disease should be explained to the patient and the possibility of arresting the condition should also be stressed Inadequate dosage of early cases is the chief reason for the bad prognosis which is so often given in cases of syphilitic aortic disease It is said by some clinicians that arsenical treatment should not be given because it is dangerous But there is no doubt that in many cases it can arrest the disease and since the lesion is usually progressive if untreated there is no logical justification for not using neosalvarsan Surgical treatment is somewhat risky in other conditions but where the chance of cure is good and where without surgery deterioration is certain an operation is willingly undertaken both by surgeon and by patient Surgeons should have no monopoly of therapeutic courage where circumstances warrant action

CHAPTER XXII

AORTIC STENOSIS

AORTIC STENOSIS is caused by rheumatism in 40 per cent and by atheromatous change in 60 per cent of the cases in the British Isles Elsewhere these proportions vary according to the climatic incidence of rheumatism Obstruction at the aortic orifice is also occasionally produced by the presence of large vegetations in malignant endocarditis, where these are implanted upon a congenital defect of the aortic valve Syphilis never causes aortic stenosis Its effect on the aortic ring is that of dilatation and not constriction

The appearance of the aortic valve in aortic stenosis is one of gross fibrous thickening and rigidity of the cusps which are frequently extensively calcified These thickened cusps are adherent at their edges, so that only in the centre is there left a small orifice for the passage of blood from the left ventricle The calcification is often sufficiently gross to be visible on the X-ray screen There is frequently an associated aortic atheroma which is curiously absent from the first few inches of the aorta next to the valve The left ventricle shows a pure concentric hypertrophy of a very marked degree, but the radiological outline of the chamber is often little enlarged above the normal It is clear from the nature of the lesion that the aortic valve cusps are no longer able to close during diastole so that some slight aortic regurgitation invariably occurs This is frequently insufficient to produce a murmur, and nearly always is not enough to produce in the circulation the changes characteristic of aortic regurgitation Therefore the physical signs are those of aortic stenosis, sometimes with the addition of a diastolic aortic murmur

Symptoms

For many years symptoms may be conspicuous by their absence, and when they occur are almost always due to atheromatous disease of the coronary orifice Their nature varies In most cases there is a progressive shortness of breath and lassitude, with the usual evidence of left ventricular failure In a few cases infarction of the coronary circulation occurs, in some coronary atheroma causes angina

of effort, and in others sudden death is the first manifestation of heart disease in these patients Syncope is not very uncommon, and may be the result of an abnormality of the carotid sinus function It is often provoked by exertion

Signs

The physical signs are characteristic, and it can be stated that aortic stenosis is diagnosed by palpation The information which can be obtained in other ways is confirmatory On inspection, the apex beat may be seen to be unusually forcible, sometimes rather diffuse On palpation, the apex beat has a firm persistent thrust, but in many cases it is very little displaced The displacement is more downwards than outwards, for much dilatation is rare Palpation at the aortic base, or a little lower, reveals the presence of a systolic thrill, which can be traced up into the vessels of the neck This thrill may be difficult to feel when the patient is lying flat but it becomes more easily distinguishable when the patient sits up and the upper chest is held firmly braced between both of the examiner s hands The pulse is characteristic and frequently appears to have a double impulse If the pressure of the fingers on the radial or brachial artery is slowly loosened and slowly tightened, a pressure can be found at which this double wave is appreciable in many cases A pulse tracing shows that the pulse wave is prolonged the so called "pulsus tardus ' The double thrust of the pulse may be explained as follows At the first contraction of the left ventricle a jet of blood quickly shoots into the aorta and raises the pressure in the peripheral arteries but it is not until a perceptible fraction of time after this that the full mass of the ventricular muscle comes into action to squeeze the full load of blood past the stenosed orifice, and it is only as a result of this final systolic effort that the second and larger wave of the pulse is produced

Percussion may reveal that the left side of the heart is slightly enlarged Auscultation shows that the first sound is accentuated and booming, and that a systolic murmur is present at the apex This murmur is loudest at the aortic base, and is also conducted into the vessels of the neck The aortic second sound is generally absent If a second heart sound is heard at the aortic base, it is probably the pulmonary second sound which has been conducted

The heart rate in aortic stenosis is frequently not increased It may be reduced below the normal figure, and the blood pressure shows a systolic reading which is not raised although the diastolic is frequently high, a typical figure being 140/110 When the left

ventricle begins to weaken the pulse pressure may be still further diminished

It should be remembered that, although the diagnosis is easy in a fully developed case, the physical signs may be very greatly diminished, and may even disappear if left ventricular failure of a severe degree supervenes In these circumstances the thrill is absent, the heart sounds become weak, and the systolic murmur at the aortic base may no longer be audible This state of affairs persists while the heart failure is severe The lesion should always be suspected when heart failure occurs with a regular and relatively slow heart rate, and with a small pulse pressure, in an elderly individual

Differential Diagnosis

Aortic stenosis must be distinguished from syphilitic or atheromatous disease of the aortic valves associated with a double aortic murmur In these latter cases in addition to the physical signs of aortic regurgitation, there may be a loud systolic murmur frequently accompanied by a thrill at the aortic base This is caused by the fact that scarring or distortion of an aortic valve cusp may cause it to protrude into the blood stream during ventricular systole This may set up sufficiently coarse vibrations to produce both a murmur and a thrill But the aortic ring is actually wider than normal, so that the murmur and thrill are isolated incidents and produce no changes in the blood pressure or the pulse wave It is the aortic regurgitation which then dominates the picture with the water-hammer pulse and the low diastolic blood pressure Aortic stenosis may have to be distinguished also from an aortic aneurysm so far as the presence of a systolic murmur and thrill are concerned But in aneurysm of the aorta, not involving the aortic ring the aortic second sound is loud and ringing There is no hypertrophy of the left ventricle and no characteristic pulse Furthermore, other signs and symptoms suggesting pressure by the aneurysm on neighbouring structures are generally present

Pericarditis may produce a systolic and diastolic sound at the base and these may be due to friction sufficiently coarse to be felt but no systolic murmur is conducted into the neck, there is no slow pulse, and other signs and symptoms of acute rheumatism are generally present

There is no treatment for the stenosis as such For many years the left ventricle is adequate to the work imposed upon it When the coronary nutrition fails, from involvement of the orifices in the

atheromatous process the patient's activities must be curtailed to suit the powers of the deteriorating heart Death is apt to be unexpectedly sudden

Subaortic Stenosis

Subaortic stenosis is a rare congenital form of narrowing of that part of the left ventricle just below the aortic ring The signs are those of aortic stenosis, but an aortic second sound may be heard, for the cusps are normal Diagnosis chiefly depends upon the discovery of the lesion in a child too young to have had time to develop aortic stenosis Symptoms are absent The prognosis is good

CHAPTER XXIII

DISEASE OF THE TRICUSPID VALVE

THE tricuspid valve may be stretched by a dilating right ventricle or it can be distorted by chronic fibrotic rheumatic valvulitis

Atonic tricuspid valvular dilatation is a common sequela of failure of the right ventricle Usually such failure is due either to mitral stenosis with persistent pulmonary congestion, or to left ventricular weakness The former is the commoner cause for failure of a severe degree is more compatible with prolonged survival in chronic rheumatic carditis, than it is in chronic myocardial degeneration primarily affecting the left ventricle In the failing heart, from either cause, there is no great hypertrophy of the right ventricle, and the valve cusps remain normally thin and flexible Thus, although evidence of right ventricular hypertrophy may be present, this does not arrest the attention

The sign of greatest value in all cases of tricuspid regurgitation is a slow expansible pulsation of the neck veins These are enlarged, the increased venous systolic pressure being manifested by obvious pulsation of the distended vessels The pulsation is easily seen, and is palpable to delicate touch A systolic murmur at the lower end of the sternum may or may not be present and is of little diagnostic value

In addition, there is often marked enlargement of the liver, and this organ too may pulsate In a thin individual the liver may be palpated bimanually and the pulsation is then felt to be expansile Other signs of severe congestive failure usually coexist

The mechanical effect of tricuspid regurgitation is to some extent that of preventing the right heart from being overburdened, so that previously severe pulmonary congestion may be lessened, with a diminution in dyspnœa and in orthopnœa

The manifestations of the causative mitral or left ventricular disease are superadded to the clinical picture

ORGANIC TRICUSPID VALVULITIS

This is nearly always secondary to chronic mitral disease, usually stenosis, and like the latter is caused by recurrent rheumatism The ætiology is identical, therefore, with that of mitral stenosis, but it is a later complication, so that the degree of the mitral deformity is

usually in advance of that of the tricuspid valve As in mitral disease, the fibrotic defect may produce either regurgitation or stenosis or both, according to the shape and capacity of the stiff valve orifice

Organic tricuspid regurgitation is shown by enlargement and pulsation of the neck veins and liver, as already described in atonic tricuspid insufficiency But, since the leak is due to a rigidly deformed valve, and not dependent upon the lack of muscular tone only it is more permanent In addition, evidence of right ventricular hypertrophy is more constant and a systolic murmur ascribable to the lesion is *more likely to be heard at the lower end of the sternum* than in the atonic variety

Tricuspid stenosis

Tricuspid stenosis is associated with a still more constant degree of venous and hepatic distension The neck veins may be grossly swollen especially on the right side The effect of tricuspid stenosis is to cause an accumulation of blood behind the valve and a deficiency in front Pulmonary congestion and, therefore dyspnœa and orthopnœa are all strikingly small in a patient with well-marked systemic congestive failure

The **signs** of tricuspid stenosis are primarily those of persistent engorgement of the neck veins, liver, and systemic circulation Pulsation in the veins may or may not be present If the rhythm is normal the chief venous pulsation, and that of the liver may be obviously presystolic If the auricles are paralysed by fibrillation the picture is that of distension only, without much pulsation In rare cases *the right auricular systole may produce a presystolic murmur* at the lower end of the sternum distinct from the presystolic mitral murmur at the apex A corresponding diastolic murmur is a theoretical possibility

The X-ray picture may be very suggestive It may reveal an exaggerated superior vena cava shadow at the upper right edge of the heart shadow, extending towards the right side of the neck Simultaneously the pulmonary fields may be unexpectedly clear, being saved from congestion because the narrowed tricuspid valve forbids excessive entry of the blood into the lungs

The patient should be treated in the usual way for the associated congestive failure, and for auricular fibrillation when this is present The prognosis may be unexpectedly good as regards life, for the lesion spares the myocardium so that a patient may live a number of years, but at a greatly reduced exercise-level

CHAPTER XXIV

CONGENITAL HEART DISEASE

CONGENITAL cardiovascular malformations are dangerous to health in two ways either the abnormality interferes mechanically with the circulation, or it offers a nidus for a secondary infection The malformation may cause deviation of the blood stream, so that a considerable fraction of the venous blood does not pass through the lungs, but is shunted to the arterial circulation, and so produces cyanosis Other varieties of malformation may or may not cause mechanical embarrassment to the efficiency of the heart Thus, there are two main clinical groups of lesions, those with cyanosis and those free from cyanosis A third intermediate group in which cyanosis may or may not occur is also recognisable

The cyanotic group is more serious both from the effect upon the duration of life and in the severity of the symptoms, but in this group secondary infection is much rarer than in the acyanotic group The cyanotic group is most frequently characterised by pulmonary stenosis This lesion is associated with other gross defects The commonest combination is "Fallot s tetralogy" But the exact determination of finer diagnostic points is often impossible in the cyanotic group

The acyanotic group comprises bicuspid aortic valve patent interventricular septum, and the vascular defect patent ductus arteriosus The commonest example of the intermediate group is atrial septal defect

CYANOTIC GROUP

The cyanosis is caused partly by a shunt of the venous blood into the arterial circulation, and partly by an increase in the amount of oxygen removed by the body tissues from the peripheral blood The peripheral circulation is appreciably slower than normal, for the peripheral blood vessels are very much dilated This slower flow is a contributory cause of the cyanosis Moreover the blood volume is increased, and this change runs parallel with the slowed circulation rate Finally there is much compensatory erythræmia, the red blood cell count and hæmoglobin being much increased

From birth the child's tissues adjust themselves to the abnormally low oxygen saturation of the blood, and the result is frequently that the body seems to work normally, without shortness of breath or other signs of distress, at an oxygen saturation of as low as 70 per cent of normal Such a low figure in a normal individual, produced by congestive heart failure, would be associated with very gross symptoms

The commonest mechanical cause of the cyanosis is pulmonary stenosis associated with a shunt of blood from the venous to the arterial side of the heart Often this shunt is through a patent interventricular septum and mixed arterial and venous blood passes up into an aorta so dextraposed as to bring its opening immediately above the defect in the ventricular septum

This general state of affairs has further consequences The right ventricle is hypertrophied, this change being associated with well-marked right axis deviation in the cardiogram, the pulmonary stenosis reduces the pulmonary blood flow, so that there is a marked absence of pulmonary congestion, radiologically, the aorta may be seen placed abnormally to the right and the pulmonary artery or pulmonary conus shadow is frequently smaller than normal

TETRALOGY OF FALLOT

The tetralogy of Fallot is a condition in which pulmonary stenosis, interventricular septal defect, dextrapostion of the aorta and right ventricular hypertrophy are all present

The syndrome may be diagnosed with greater confidence than any other form of cyanotic congenital heart disease, partly by the signs and symptoms and partly because it is much commoner than the other forms in children who survive

Symptoms

The chief symptoms are two varieties of dyspnœa Shortness of breath is present with exertion to an abnormal extent, but less than would be expected from the amount of obvious cyanosis A *second variety of dyspnœa is that which comes on in sudden severe* attacks, bearing a slight resemblance to Stokes-Adams attacks The patient is seized with increasing dyspnœa, increasing cyanosis, and a state of asphyxia in which convulsions not infrequently occur These attacks may be fatal It is usually considered that they are a direct result of erythræmia, and that the site of the trouble is in

the brain Other symptoms are epistaxis and bleeding from the lungs or even the stomach

Signs

The physical signs are as follows cyanosis is always present and is associated with an increase in the blood count sometimes to the unusual figure of 8 to 10 million red blood cells with a corresponding increase in the hæmoglobin percentage Clubbing of the fingers

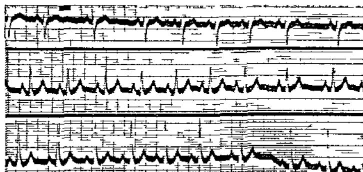


FIG 45 FALLOT'S TETRALOGY

Th

is generally seen but may be absent under the age of two Examination of the heart shows evidence of hypertrophy of the right ventricle whose heaving impulse is often palpable in the epigastrium A systolic thrill and a loud systolic murmur are both present generally over the second and third left interspaces near the sternum The murmur is harsh and is conducted up into the carotids probably by way of the dextraposed aorta and its branches The absence of this conduction into the neck vessels is a diagnostic point against the presence of an associated ventricular septal defect In some cases the thrill may be absent and the murmur may be distant or absent here the pulmonary artery is a narrowed tube rather than a locally constricted one and the ventricular septal defect is large Both of these characteristics would militate against the production of a thrill and would cause the murmur if present to be softened The patient has a curious predilection for the squatting position

The electrocardiogram (Fig 45) always shows well marked right

axis deviation. The most typical X-ray changes (Fig. 46) are that the pulmonary conus area is concave instead of convex, that the aorta is displaced slightly to the right, and that the ventricular shadow is prominent by contrast, so as to produce a boot-shaped



Fig. 46.—Fallot's Tetralogy

The heart is enlarged (sabot-shaped). Note especially the absence of pulmonary conus shadow. There is no aortic knuckle, the aorta being dextraposed. Boy aged 5 years.

heart. But in many cases there is slight general enlargement without a sabot heart. Pulmonary congestion is markedly absent.

The most common complications are cerebral thrombosis, or possibly hæmorrhage, and pulmonary tuberculosis.

Operative Treatment

Operative treatment of this condition has recently been introduced. It consists in forming an anastomosis between the subclavian or

the innominate artery on the one hand and a large branch of the pulmonary arterial circulation on the other This procedure permits full oxygenation of a considerable fraction of the arterial blood to occur In properly selected cases the operation may lead to excellent results Accurate diagnosis is essential The operative mortality is still considerable being in the neighbourhood of 15 to 20 per cent

Prognosis

Without operation the outlook as regards survival is poor, the average mortality occurring between the ages of five and ten When the lesion is small in degree patients live sometimes into the twenties or thirties, very rarely even to late middle life

ACYANOTIC GROUP

BICUSPID AORTIC VALVE

The aortic valve may have two cusps either as an isolated abnormality, or in association with some other congenital defect usually also of the acyanotic variety The commonest associated abnormalities are coarctation of the aorta and patent ductus arteriosus *A bicuspid aortic valve is functionally as efficient as a normal* valve with three cusps There is usually no leak and therefore no murmurs or organic changes in the heart, blood pressure or circulation

Infection of a bicuspid aortic valve, however is common, and when this happens the valve becomes defective so that aortic regurgitation occurs This is associated with fever toxaemia, signs of embolism, and a positive blood culture It constitutes one of the varieties of subacute bacterial endocarditis When aortic regurgitation with fever develops in a patient, whose heart was previously normal on examination, it is highly probable that the cause of the illness is infection of a bicuspid aortic valve The same conclusion can be drawn when aortic regurgitation with fever develops in an individual known to have a patent ductus arteriosus and ventricular septal defect or a coarctation of the aorta The diastolic murmur produced by the leak is apt to be misleading when it is associated with a systolic murmur due for instance, to a patent interventricular septum The production of this double murmur can fallaciously suggest the diagnosis of patent ductus arteriosus

The **physical signs** of the aortic leak and the characteristics of the secondary infection are identical with those of aortic regurgitation due to other causes and to subacute bacterial endocarditis imposed upon organically acquired valvular disease

PATENT INTERVENTRICULAR SEPTUM

This condition may occur alone or it may coexist with other congenital abnormalities When it occurs alone it is sometimes known as Roger s disease When it occurs with other conditions such as Fallot s tetralogy it plays a minor part in the symptomatology There is one exception to this statement and that is when the defect is so large as to constitute the condition of trilocular heart

When the patency occurs alone it is usually situated at the posterior part of the anterior septum when it occurs in association with other congenital abnormalities it is usually found at the anterior part of the anterior septum

Patent interventricular septum as an isolated lesion or Roger s disease is common It is usually diagnosed easily in childhood if the heart is examined because the murmur produced is loud and harsh

The effects on the heart are that there is a leak generally small from the left ventricle to the right for the left ventricular pressure is higher Since this merely introduces a little arterial blood into the pulmonary circulation there is obviously no cyanosis The left ventricle is hypertrophied in proportion to the extent of the leak There is usually no dilatation The lesion clearly also imposes a small added burden on the right ventricle and a proportional increase of right ventricular muscle probably occurs in all cases

There are no **symptoms** unless subacute bacterial endocarditis occurs or unless long possession of a loud murmur has caused the patient to develop a cardiac neurosis as a result of the warnings of a succession of over anxious practitioners The physical signs are those of hypertrophy of the left ventricle which although little displaced becomes more forcible and heaving In addition there is a systolic thrill and murmur best elicited in the third and fourth spaces near the left border of the sternum The murmur is transmitted to some extent to the pulmonary base but not into the axilla The blood pressure is normal The X ray shows a rather rounded left ventricle but a normal pulmonary conus The electrocardio-

gram is usually normal, although in some cases there may be a little left-axis deviation

The prognosis is good in the case of a small lesion Occasionally, in babies and in small children, the defect seems to close, since the physical signs may disappear as the body develops In the average case the risk of endocarditis is always present, and it is probably by this means or by a lessened power to withstand wear and tear that the outlook as regards length of life is shortened It is unusual to come across cases older than forty five

The **differential diagnosis** is generally easy In patent ductus arteriosus the murmur extends into diastole and both murmur and thrill are best heard in the second intercostal space It must be remembered that, as a rare occurrence, a myocardial infarct may perforate the septum, and give rise to the typical thrill and murmur The history should enable this situation to be cleared up Congenital heart block occasionally coexists with a patent interventricular septum

PATENT DUCTUS ARTERIOSUS

When the ductus arteriosus remains patent a leak is thereby introduced from the aorta into the pulmonary artery This allows blood to flow from the aorta into the pulmonary artery both in systole and in diastole

This lesion is usually found uncomplicated by other abnormalities, but when these occur with it they are likely to be either a bicuspid aortic valve or coarctation of the aorta Although strictly speaking the lesion is a vascular defect, it is useful to consider it with the congenital cardiac abnormalities proper because of the associated changes in the heart and circulation

These are, in short, the usual adjustments produced by any form of leak from the aorta, the commonest example of which is aortic regurgitation As in this latter lesion both systole and diastole are affected The left ventricle has more work to do during systole and is partially deprived of the help it usually receives from the force stored in the elastic recoil of the aorta This results in both hyper trophy and in some dilatation The leak into the pulmonary artery, by somewhat increasing the pulmonary pressure, also produces a proportional hypertrophy of the right ventricle The diastolic blood pressure is lowered by the leak The systolic pressure may be slightly raised Thus the pulse pressure is increased in proportion to the size of the patent ductus There is an effect also on the general state of health, so that a child may fail to grow normally

Symptoms

In many cases the lesion itself produces no symptoms, but in others, if the leak is large, shortness of breath and general physical deficiency become apparent Less often, particularly in older patients, and if the leak is considerable, congestive failure with œdema and ascites may occur Finally, infection of the ductus, as in subacute bacterial endocarditis is fairly commom

Physical Signs

The left ventricle is hypertrophied and dilated in proportion to the extent of the leak If this is small there is no enlargement, but if the lesion is large the increase in size of the left venticle may be very considerable The most typical signs are the thrill and the murmur characteristic of the defect These are localised chiefly in the second space and less commonly, behind the third rib, and if transmitted at all are conducted towards the lower border of the inner half of the left clavicle But the murmur and thrill may be so localised as to be missed unless the stethoscope is placed exactly over the proper position The murmur is systolic, and in its typical form is carried well into diastole It may start with the beginning of systole and may then increase in loudness during later systole This is not characteristic of most other systolic murmurs, which are loudest at their onset It may be easily audible or may be very distant especially in adults It may be high-pitched and blowing, but usually *has the well-known machinery sound It may suggest* the revolving clash of distant machines or it may be rumbling and reminiscent of the Scottish express running downhill in the tunnel outside King's Cross with the brakes off the rhythmic waxing and the waning of the roaring sound being almost exactly reproduced In rare cases the murmur is systolic only The thrill generally accompanies the murmur and varies with it The blood pressure change is proportional to the size of the leak, and the lowered diastolic pressure is particularly definite after drastic exercise, this is sometimes a valuable method of confirming the diagnosis

Usually the electrocardiogram is normal although some left-axis deviation may occur The X-ray (Fig 47) is most valuable as a method of diagnosis, for it usually shows the typically prominent pulmonary conus, which sits like a little cap on the upper border of the left cardiac outline If the left ventricle is much enlarged this prominence may be almost completely obscured In the right

oblique position also the pulmonary conus is visible in the upper part of the anterior outline. The screen reveals considerable pulsation in the enlarged conus. The pulmonary artery shadows are generally prominent on the film.



FIG. 47.—PATENT DUCTUS ARTERIOSUS

Showing the typical bulge in the region of the pulmonary conus. There is slight general enlargement of the heart shadow. Boy aged 9 years.

Diagnosis

In the presence of the typical murmur and thrill the diagnosis is easy, and if the leak is sufficient to affect the blood pressure considerably there can be no doubt. The association of systolic and diastolic murmurs may occasionally give rise to difficulty, one example of this being the association of an interventricular septal defect with an infected bicuspid aortic valve. Another cause of difficulty, especially in children, is an unusually loud venous hum in

the jugular veins, this may also cause a loud, rather low-pitched systolic murmur, which passes on into diastole, and in some cases is almost continuous This murmur is easily diagnosed, since it can be stopped by pressing firmly over the internal jugular vein just above the clavicle When a systolic murmur alone is present, it is necessary to remember the possible presence of simple pulmonary stenosis, but here the X ray picture shows a pulmonary conus and pulmonary vascular shadows less definite, not more definite, than normal Occasionally some difficulty is experienced in separating patent ductus arteriosus from interventricular septal defect, especially if the heart is enlarged In cases of interventricular septal defect there is no increase in the pulmonary conus, and the blood pressure is normal *It is probably true that in most cases where real doubt* exists the lesion is not that of patent ductus arteriosus

Infection of the ductus produces fever, lassitude, sweating, and evidence of embolism as in other cases of subacute bacterial cardiovascular infection The lungs are the usual receptacle of the emboli, so that transient cough, pleurisy, or areas of congestion are found A positive blood culture may be difficult to obtain in some cases, since much of the infected blood, if not all of it, passes through the lungs, and the organisms are filtered off there

Treatment

The patent ductus can now be satisfactorily tied The chief indications for advising this are failure of the child to grow, symptoms of shortness of breath or cardiac disability, the presence of cardiac enlargement, congestive failure, and the fact that so few cases of patent ductus arteriosus over forty years of age come under observation in consulting practice, whereas many are seen in the younger age groups, the implication being that they do not survive When the ductus becomes infected the infection also may be cured by surgical ligation, but it is probably wiser to treat the septicæmia first by penicillin, as in subacute bacterial endocarditis

POTENTIALLY CYANOTIC GROUP

INTERAURICULAR SEPTAL DEFECT

The auricular septum may be deficient either because of the persistence of the normal foramen ovale, or as a result of gross maldevelopment of the interauricular septum Patency of the

normal foramen ovale is a harmless curiosity, giving rise to no symptoms or signs, or subsequent troubles Gross defect of the auricular septum is most usually seen in the form of a persistent ostium primum, producing a large patency just above the auriculo ventricular valves

The changes in the circulation caused by this abnormality are best realised when it is remembered that the left auricle is situated above and the right auricle below, when the body is in the upright position During auricular diastole the auricles constitute two rather thin-walled bags containing blood connected with one another through the patency It is clear that gravity is likely to cause some continuous flow of blood from left auricle to right auricle through the abnormal opening The volume of blood flowing around the pulmonary circulation will thus be progressively increased over the course of years

This increasing pulmonary congestion and plethora constitute the main characteristics of the lesion The pulmonary arteries become greatly enlarged, so as to be prominent in the X ray picture The pulmonary valve may become so stretched as to be incompetent, giving rise to pulmonary regurgitation The auricles particularly the right auricle, become also progressively enlarged and there is also some enlargement of the right ventricle Simultaneously there is an ischæmia of the systemic circulation, so that the aorta is hypoplastic and general physical development may be somewhat retarded The actual circulation pulmonary and systemic, is adequate for ordinary activities, so that cyanosis and failure do not usually occur except as a result of overstrain, pulmonary complications, or intercurrent acquired heart disease

Symptoms are frequently absent until adolescence or adult life The frame is sometimes rather poorly developed and actual cases of infantilism have been observed Heart failure of the congestive variety may ultimately supervene, and the symptoms are then typical of this

The **physical signs** are prominent There is no cyanosis or clubbing of the fingers as a rule, but these may appear later in life Although the heart is clearly much enlarged, the actual apex beat is not thrusting or strong, because the right ventricular enlargement rotates the left ventricle backwards away from the chest wall The heart is enlarged to the right, as shown by percussion A typical systolic thrill and murmur are usually present to the left of the sternum in the third or fourth spaces, and pulsation from the enlarged pulmonary conus is frequently palpable in the second space to the

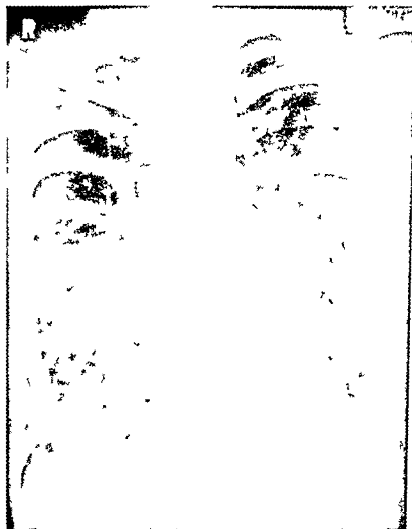


Fig. 48 Interventricular Septal Defect

Transverse cardiac diameter 15.0 cm. There is general enlargement of the heart and considerable enlargement of the pulmonary conus. The mass ... hilar shadow is due to a grossly dilated right pulmonary artery. The left pulmonary artery was found equally large at the post mortem. Adult aged 33 years

left The murmur is blowing and definite in character The pulmonary second sound may be remarkably accentuated, especially in children, and in some cases the diastolic murmur of pulmonary incompetence may be audible over the pulmonary area and in the second or third left space near the sternum This murmur may be heard at any stage in the disease, and is probably due to an additional congenital abnormality of the pulmonary artery or the pulmonary valve There are no associated signs of aortic regurgitation, such as arterial pulsation or a lowered diastolic blood pressure

X-ray examination shows a large, frequently a very large heart, globular in general outline but with obvious prominence of the pulmonary conus, and an insignificant aortic knuckle (Fig 48) The two pulmonary arteries sweep outwards and downwards the right one often in the form of a huge comma, the left partially obscured by the heart shadow Screening examination often shows pulsation in these big vessels, especially if pulmonary regurgitation is also present

The electrocardiogram rather paradoxically may show a normal axis deviation, although some slight right axis deviation may occur Auricular hypertrophy may produce prominent P waves

The chief cardiac complication of the lesion is rheumatic mitral stenosis, this picture going by the name of Lutembacher's syndrome Mongolianism sometimes coexists Infective endocarditis is rare

This form of congenital heart disease is the only one in which rheumatic heart disease is a frequent complication The subsequent mitral stenosis may cause auricular fibrillation This irregularity is rare in other forms of congenital heart disease When it occurs, *œdema of the legs and feet commonly follow and the ascites and liver* enlargement are often increased out of proportion to the rest of the congestive picture

The **prognosis** is fairly good, the average expectation of life in some recorded series of cases being as long as thirty-six years Survival into the sixties has occasionally been noted

The **differential diagnosis** is usually straightforward, but difficulty may arise in distinguishing between this condition and advanced cases of simple acquired mitral stenosis, particularly if no thrill or murmur is present over the pulmonary area In mitral stenosis the X-ray is likely to show more widespread pulmonary congestion, in patent interauricular septum it is likely to show very well defined and large pulmonary arteries

OTHER VARIETIES OF ACYANOTIC CONGENITAL HEART DISEASE

The Eisenmenger complex is one in which there is a large interventricular septal defect, with dextraposition of the aorta, and dilatation of the pulmonary artery. The heart is rather globular on the X-ray film, with a prominent pulmonary conus but without the great enlargement of the pulmonary arteries seen in interauricular septal defect. The electrocardiogram shows right axis deviation.

There are several varieties of the trilocular type of congenital heart disease, the one in which survival is most possible is that in which the interventricular septum fails to develop, the ventricles constituting a single chamber; the auricles remain normal. This is often called the cor triloculare biatriatum. Dextraposition of the aorta is usual in the cases which survive.

In both of these cardiac malformations cyanosis is absent at first, but may develop later. *Cyanosis and clubbing are more prominent* in the trilocular heart and occur earlier in life than in Eisenmenger's complex. The diagnosis of these and similar conditions is a matter of difficulty and requires careful special study in each individual case.

COARCTATION OF THE AORTA

Congenital narrowing of the aorta is generally described as occurring in two ways. There is a tubular narrowing, which is incompatible with prolonged life, so that the infant does not survive, and there is a local constriction of varying extent which is generally not discovered until later childhood or adult life. The cause of the local constriction is unknown, but it is thought that it is caused by abnormal contracture of plain muscle fibres in the aorta. These muscle fibres may be similar to other muscle fibres normally present in the ductus arteriosus, and which are responsible for its closure soon after birth. If some of these were misplaced, being laid down in the aorta by congenital mal-development, they could well be responsible for the local coarctation.

Several points *are of significance: coarctation of this type occurs* in the aorta at the level of the origin of the normal ductus arteriosus, patent ductus arteriosus and coarctation coexist fairly often, both lesions being the result, presumably, of abnormalities of position and function of the above mentioned muscle fibres, subacute bacterial endarteritis, caused by the streptococcus viridans, may complicate

patent ductus arteriosus and coarctation, either separately or together

The **signs** of coarctation of the aorta are as follows The local narrowing, which is just distal to the left subclavian artery, causes a great increase in the blood pressure of the arms, head, and neck and a corresponding lowering of the pressure in the legs This *added burden brings about left ventricular hypertrophy* which is not associated with dilatation so long as the ventricles are healthy The increased force of the blood pressure in the upper parts of the body stimulates collateral circulation towards the lower parts where the blood pressure is low, chiefly by means of the mammary arteries and the scapular, axillary and superior epigastric arteries

There are no **symptoms** unless the condition is complicated by other pathological conditions and it is usually discovered accidentally The physical signs are those of left ventricular hypertrophy associated with an increase in the brachial systolic blood pressure, figures of 150 to 200 millimetres of mercury may be found in the brachial arteries The readings may vary in the two arms In the legs a lowered systolic blood pressure of, for example, 50 to 120 millimetres of mercury is discovered the pressure varying according to the degree of narrowing It is also noticeable on palpating the abdomen and the common iliac vessels that pulsation is *very weak or absent* *The arms and upper chest may feel distinctly* warmer to the touch than do the legs and feet In some cases severe epistaxis may suggest the diagnosis, or the symptoms of intermittent claudication in a young individual may arouse suspicion as to the possible cause

The method of taking the blood pressure in the legs is as follows if there is an easily palpable dorsalis pedis artery, or posterior tibial artery, the armlet of the sphygmomanometer can be applied firmly around the lower leg and the systolic blood pressure taken by palpation in the usual way Alternatively, with the patient lying on his side, the armlet is wrapped around the thigh just above the *popliteal fossa, taking care that the centre of the air reservoir lies* over the lower part of the femoral artery The armlet may have to be reinforced by a piece of broad bandage to prevent slipping or distension The stethoscope is then placed over the popliteal artery and the pressure can be taken as usual by auscultation

The superficial arteries of the thorax enlarge considerably so that they are usually visible by careful inspection The patient should be placed in a good oblique light The regions particularly looked at are the interscapular, lower dorsal, and axillary areas The

patient is slowly rotated so that the pulsating vessel is well illuminated and is contrasted with the more shadowy background. Pulsation in these enlarged vessels is then easily visible and can be confirmed by palpation. The vessels are often made prominent if the patient

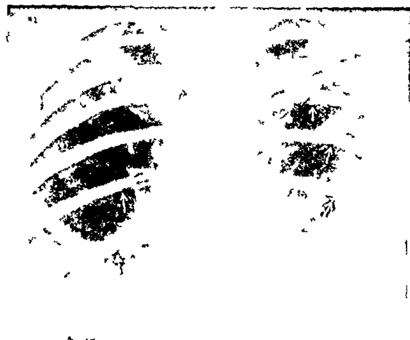


FIG. 49.—COARCTATION OF THE AORTA

There is marked notching of the lower margins of the ribs, especially of the fifth, sixth and seventh on the left and of the sixth and seventh on the right side. The aortic knuckle is strikingly small. The left ventricle is enlarged.

stands with straight knees and loosely hangs his chest and arms forwards and downwards towards his toes.

In addition to this evidence of circulatory change the constriction in the aorta gives rise to a systolic murmur, frequently rather harsh and often quite loud. The murmur is analogous to that of aortic stenosis in its general qualities and comes in a rather prolonged crescendo, not reaching its loudest until a measurable fraction of time after the start of systole. But it is more distant than the murmur of aortic stenosis since the lesion is deeper in the chest and its position of loudest intensity is usually an inch or two to the

left of the sternum in the third or fourth space From the position it may be conducted to almost any part of the thorax, but most readily to the posterior scapular and lower dorsal areas

X-ray examination (Fig 49) shows some enlargement of the left ventricle due to hypertrophy this enlargement increasing if the heart is dilated from intercurrent myocardial degeneration, or from aortic regurgitation due to stretching of the aorta The ascending aorta is usually clearly visible, but the aortic knuckle is generally small The ribs show characteristic erosion from pressure of the intercostal arteries, along their lower borders This erosion may be in the nature of notches, or may suggest longer, whittled depressions These changes are roughly symmetrical and bilateral The electro cardiogram is usually normal, but may show some left axis deviation

Presumably complications are due chiefly to the onset of degenerative changes in the unusually strained aorta Aortic incompetence may occur and may produce left ventricular failure The aorta may rupture completely causing sudden death or partially, causing a *dissecting aneurysm, which in its turn is fatal after a short time* Cerebral hæmorrhage is not uncommon either from the development of atheroma, or from the presence of congenital aneurysms which may also occur in these patients The site of the narrowing may become infected by the streptococcus viridans causing a febrile illness **Treatment** of this is by penicillin as in infective endocarditis Surgery, in appropriate non-infected cases is beginning to give successful results

CHAPTER XXX

MALIGNANT ENDOCARDITIS

THERE are two varieties of this condition acute infective endo carditis and subacute bacterial endocarditis They have certain characteristics in common but their clinical course differs

In each there is infection of a previously diseased valve or of a congenital cardiac or vascular defect In each fever bacteriæmia cardiac murmurs and signs of embolism are found In acute in fective endocarditis the organism may be almost any of the pyogenic bacteria and the course of the disease is restricted to days or weeks On the other hand in more than 90 per cent of cases of subacute bacterial endocarditis the infective organism is the streptococcus viridans and the length of the disease may be many months or even a year or more

ACUTE INFECTIVE ENDOCARDITIS

The infective organism varies it is most often either the strepto coccus hæmolyticus or a staphylococcus the B coli or the pneumo-coccus The valve infection usually derives from an acute strepto-coccal or staphylococcal infection elsewhere in the body or from a pneumonia It may coincide with the primary condition or it may closely follow it The organism reaches the heart valve cusps upon which rather large shaggy vegetations develop The left side of the heart is more often affected than the right but the right side is more commonly attacked in acute endocarditis than in the subacute bacterial variety

The blood stream infection and the associated toxæmia dominate the clinical picture and the cardiac lesion may be missed unless it is revealed by careful physical examination or deduced from the occurrence of gross embolism of the spleen brain or other organs The presence of purpuric and petechial spots is found of course equally commonly in uncomplicated septicæmia The **symptoms** are those of an acute general infection with prostration sweating rigors and a quickly developing secondary anæmia

The cardiac murmurs most frequently detected are those of

mitral regurgitation, aortic regurgitation, and congenital interventricular defect, but murmurs characteristic of any acquired or congenital heart lesion may be present. Infection of minor pulmonary valve defects is especially difficult to diagnose, for the systolic *pulmonary murmur is often regarded as hæmic or functional*, and there may be no thrill.

The gross embolic phenomena are identical with those of subacute bacterial endocarditis, but the petechiæ and purpuric spots may differ in some respects. *These are often remarkable for the* speed with which they appear, the increasing rash seeming to come out almost visibly from hour to hour, or from day to day. The individual petechia is often larger than that seen in subacute bacterial endocarditis, and the centre may be slightly discoloured. Pulmonary embolism, small in extent, is not uncommon in infections of the right side of the heart, showing itself by recurrent attacks of slight chest pain and cough, with fine localised pleurisy, and occasionally by a little hæmoptysis.

The blood culture is generally positive, but it may be necessary to repeat it several times. If it is negative, seeming to contradict the clinical picture, the hydrogen-ion concentration of the broth may be at fault. This must be at, or near to, a figure of pH 7.6. Infection of the right side of the heart, *especially if the pneumococcus is the* infecting organism, may be slow to reveal itself by a positive blood culture, perhaps owing to the filtration effect of the pulmonary circulation.

The **differential diagnosis** lies between septicæmia without a cardiac infection, typhoid fever, meningitis, and acute lymphadenoma. In all cases of septicæmia the heart should be carefully examined at intervals, and any change in physical signs noted.

The fever, headache, prostration and splenic enlargement may suggest typhoid. But in typhoid the heart rate is low in proportion to the degree of fever, the rash is not purpuric, and there is a leucopenia; in acute endocarditis the heart rate is raised, the rash is purpuric, and a leucocytosis occurs in all but fulminating infections. The Widal reaction, repeated if necessary to show an increase in the titre of the agglutinins, together with bacteriological examination of blood, stools, and urine, should clarify the position. Meningitis, from the presence of meningeal embolism, may occur in endocarditis ... raction ... n, may ... organic cardiac murmur and the character of the cerebrospinal fluid should

elucidate the problem Acute lymphadenoma may cause an acute febrile illness with rigors and splenic enlargement, simulating both typhoid fever and acute endocarditis In such cases the heart may also show a systolic murmur The absence of other embolic signs and the negative blood culture, will help to exclude endocarditis; eventually enlargement of superficial lymphatic glands may allow the question to be settled by biopsy

Treatment of the acute form of endocarditis does not differ from that of the subacute bacterial variety in cases where the infective agent is the staphylococcus, the streptococcus, or the pneumococcus But when the B coli, the B pyocyaneus, or the enterococcus are the cause, sulphanilamide drugs must be used in full doses, even up to 12 grammes daily, for these organisms are not sensitive to penicillin When such large doses are used particular watch must be kept for symptoms and signs of overdosage, such as agranulocytosis, rash, suppression of urine, delirium, and, after subsidence of the original fever of the infection, drug fever

SUBACUTE BACTERIAL ENDOCARDITIS

The streptococcus viridans is the causative organism in all except about 10 per cent of cases In this remaining fraction the hæmophilus influenzæ, the gonococcus, or the meningococcus are found The valve infection is invariably imposed upon some previously existing acquired or congenital defect Tooth extraction is the commonest known event causing blood-stream inoculation by the streptococcus viridans, resulting in valvulitis Here the organisms present in the mouth, or in an apical infection, are allowed abrupt access to a comparatively large vascular area in bone, from which they are absorbed into the blood stream

The **clinical manifestations** are of two varieties, according to whether they are due to septicæmia or to embolism The general symptoms are lassitude, malaise, loss of weight, and profuse sweating These are often present in a patient who has a pale sallow look Indeed, the description of pallor as a sign of aortic regurgitation probably owes its origin to cases in which that lesion has a bacterial origin Vague slight pains of a fibrositic character are often complained of in muscle or joints These logically belong in the embolic group, but are often sufficiently slight and vague in distribution to suggest a general toxæmia or "rheumatic" state rather than a local vascular lesion

The *embolic phenomena* can be subdivided into slight and severe,

according to the size of the embolus The embolic symptoms may *be the first indication of the disease, the associated toxæmia being so* slight as to be regarded only as fatigue Small emboli occur in the finger-tips, the skin, the retinæ, and the kidneys, giving rise respectively to tenderness, purpuric rash, visual disturbance, and hæmaturia The rash is a fine purpuric eruption, often sparse, coming out spasmodically over any part of the skin, but being especially visible over the chest, the forearms, and under the finger-nails, where it is often elongated and splinter-like The individual spots fade in two or three days They also should be looked for under the conjunctiva, either inside the eyelids or in the sclerotic The hæmaturia is often microscopical only It is not proved whether these small hæmorrhages are truly embolic or whether they are toxic Fresh bright red petechiæ, on the trunk, are sometimes confused with the little red hæmangiomata called "Campbell de Morgan" spots The distinction is easy, for these latter can be felt by a light stroke of the finger-tip True petechiæ are impalpable

The larger emboli produce a variety of symptoms according to the position of the infarct In the spleen they cause pain, often increased by respiration, for the peritoneal surface of the enlarged spleen is inflamed Friction can sometimes be heard over it In the brain the anatomical position determines the picture In the middle cerebral artery the embolus produces sudden hemiparesis or hemiplegia, in a surface vessel it causes meningeal symptoms, headache, vomiting, neck rigidity, and possibly paresis, in the retinal artery, blindness, in a "silent" area "I felt a sudden queer click in the head" said one patient, describing the onset of his illness Renal embolism produces lumbar discomfort and gross hæmaturia Mesenteric embolism is shown by an acute abdominal picture of sudden pain, tympanites, decreased bowel motility, and possibly by vomiting or the passage of some blood Coronary embolism may cause sudden unexpected death, as in an apparently fit young Welsh footballer who travelled to London for a match, and died suddenly in his hotel Blockage of a peripheral artery causes pain, pallor and disuse of the extremity, and if function is restored the local infection may cause a mycotic aneurysm The popliteal, the brachial, and the carotid vessels are typical sites In the muscle-pulp embolism causes swollen, tender, reddened and warm areas in the finger-tips, the thenar, or hypothenar eminences, and elsewhere These are [illegible]

A final sign worthy of mention is clubbing of the fingers The origin of it is obscure, but presumably there is some peripheral interference with adequate vascular nutrition Its presence in a febrile patient with a cardiac lesion always suggests, but its absence does not exclude, infective endocarditis

As terminal events, caused possibly by a combination of fine repeated embolism and toxæmia, congestive heart failure and uræmia are to be noted The occurrence of these after apparent cure with penicillin suggests that post-inflammatory fibrosis may be the predominant factor in their causation

Clinical pathology assists and even proves, the diagnosis A positive blood culture, especially of streptococcus viridans is practically proof of the condition, when fever and a cardiac lesion are also present But in many subsequently proved cases the blood culture may be repeatedly negative Possible reasons for this are an unsuitable pH of the broth, removal of blood during an afebrile phase, too scanty a bacteriæmia, and the presence of chemotherapeutic agents in the blood stream The investigation must therefore be repeated several times, and in favourable circumstances, if it is at first negative A secondary hypochromic anæmia is nearly constant, but the hæmoglobin may not fall below about 75 per cent or 80 Maintenance of a figure of about 90 per cent is a point against the presence of the disease A leucocytosis is usual Examination of the urine shows some red blood cells throughout the course of the disease, but as time goes by casts become more frequent With development of diffuse embolic focal nephritis there may appear evidence of renal failure in the form of a raised blood urea, and a diminishing urea clearance figure

The clinical course of the disease varies considerably The dominant picture is that of a very slowly progressive toxæmia, but this may be interrupted at any time by some serious embolic complication Such an event usually depresses the state of health to a lower level, if it does not prove fatal

Differential Diagnosis

The four cardinal points in the diagnosis are fever, a cardiac lesion, signs of embolism, and a positive blood culture Any one of these may be misleading in its particular way Although fever is nearly always present, yet occasionally afebrile remissions intervene in subacute bacterial endocarditis Fever, moreover, is so common an event that its presence in a patient with a cardiac murmur is not diagnostic of any one condition The particular cardiac lesions

most common in this disease are the regurgitations and the acyanotic congenital defects, but any organic lesion is a possible nidus The murmurs generally have the character of those proper to mitral or aortic disease, but endocarditis may be suggested if the quality of the sound undergoes rapid change, or, in the case of the aortic diastolic murmur, if the sound acquires a twanging, snoring, or musical quality, instead of the usual high pitched distant rush This new *sound suggests rupture, perforation, gross ulceration, or distortion* of a valve cusp The complete absence of all signs of an organic valvular lesion is strong evidence against a diagnosis of endocarditis

Gross systemic arterial embolism, in brain spleen, kidney, mesenteric artery, and elsewhere, is common also in simple mitral stenosis, with or without auricular fibrillation If permanent auricular fibrillation is present, the embolism is probably due to detachment of non infected auricular clot, and not to subacute bacterial endocarditis This latter condition rarely infects hearts where the auricles are fibrillating But it occasionally happens that in endocarditis the disease produces, by direct spread, infection of the wall of the left auricle Here the local irritation may set up auricular fibrillation, usually quite obviously occurring in paroxysmal attacks I have once seen embolism of the brachial artery in a man who suffered from severe femoral phlebitis during which he also had suffered four attacks of pulmonary embolism Between two of these came the attack of systemic brachial embolism Almost certainly the clot arose in the femoral vein, passed to the right auricle, then reached the left auricle through a silent patent forearm ovale and thence travelled to the arm This is an example of ' paradoxical embolism "

A positive blood culture, even of streptococcus viridans is not necessarily diagnostic, for this organism can reach the blood stream from an apical tooth abscess without infecting the heart which may at the same time emit a functional apical systolic murmur Nevertheless these pitfalls are rare and as a rule the picture is so typical as to cause little doubt

There are certain other diseases which may be confused with subacute bacterial endocarditis Acute rheumatism sometimes presents cardiac lesions, with continued low fever for weeks or months The spleen may be enlarged, and a rash may appear But the blood culture is persistently sterile, no other signs suggesting infarction occur in the acute phase and the rash is rarely purpuric The joint inflammation subsides immediately under adequate salicylate therapy Pericarditis, common in rheumatism, is very

rare in bacterial endocarditis Pernicious anæmia may show fever, a cardiac murmur suggesting mitral regurgitation, a petechial rash, and enlargement of the spleen The blood count shows a macro cytic anæmia with a leucopenia, not a leucocytosis Splenic anæmia may give a very similar clinical picture Here again there is a leucopenia

Another problem is presented when a patient with an organic cardiac lesion develops persistent fever Such a case was that of a man of thirty-five who had an enlarged left ventricle, with mitral stenosis and regurgitation He became ill with persistent fever, profuse sweating, joint pains, and general prostration The spleen was enlarged and a transient, undefined erythematous rash was noticed The blood culture was persistently negative After some weeks of illness it was noticed that the hæmoglobin remained at the level of 90 per cent Since this seemed incompatible with the diagnosis of subacute bacterial endocarditis, the possibility of some other cause for the fever was considered The prostration, sweating, and joint pains suggested undulant fever His blood contained agglutinins against B melitensis to a high titre, and after many weeks illness he recovered

Treatment

Until recently this disease has been without hope of cure Chemotherapy with the sulphonamide drugs shed a ray of hope, and the advent of penicillin has at last made cure a practical possi bility The dose of penicillin necessary to effect a cure is in the neighbourhood of 500 000 units to 2,000 000 units daily for twenty-eight days or even for six weeks It is administered by three-hourly injections Penicillin treatment is not uniformly successful The mortality rate still remains in the neighbourhood of 25 per cent Half of these patients die from arterial systemic embolism, for the healing valve cusps shed thrombi, which lodge in brain coronary artery, or some other vital area The remainder die of uræmia from heart failure, or from pulmonary embolism caused by stasis and thrombus formation in the leg veins Transfusion is desirable as a subsidiary method of treatment when the hæmoglobin is reduced to an appreciable extent Heparin has been added to penicillin but equally good results have been obtained without it

Section VII

VASCULAR DISEASE AND THE HEART

CHAPTER XXVI

THE EFFECTS OF ARTERIOSCLEROSIS UPON THE HEART

A HIGH percentage of all cases of heart disease met with in both hospital and private practice in all parts of the world is due to arteriosclerosis In hospital practice the percentage of cases caused by acute rheumatism is higher, but in some subtropical and tropical countries acute rheumatism is rare, so that in these places the percentage of cases of heart trouble due to arteriosclerosis is proportionately increased In Great Britain, according to the district and the type of practice, arteriosclerosis is probably responsible for from about 55 to 65 per cent of all cases

There are several distinct varieties of arteriosclerosis, and each has a different effect upon the heart, the commonest are, medial or hyperplastic sclerosis, atheroma, and Monckeberg's sclerosis

Syphilis also affects the heart through the vascular system, but it is more convenient to treat that subject separately Periarteritis nodosa is a very rare inflammatory disease of arteries, in which the coronary vessels may become involved so as to produce infarction of the myocardium

Medial Sclerosis

This is the process associated with and responsible for hypertension, or raised blood pressure Its causation and its effects are described in the chapter on hypertensive heart disease

Atheroma

This is a disease of the aorta, of the larger arteries, and of some special vessels such as the cerebral arteries and the coronaries The lesion is patchy in its distribution It begins in the deeper layer of the intima and this thickened tissue soon degenerates At first the change is hyaline, but it later becomes lipoid in character, and the deposition of calcium salts occurs Over the degenerate areas of

fatty change and of calcification the more superficial layer of the intima des being undermined In this way atheromatous ulcers are formed The aorta is nearly always affected to some extent in

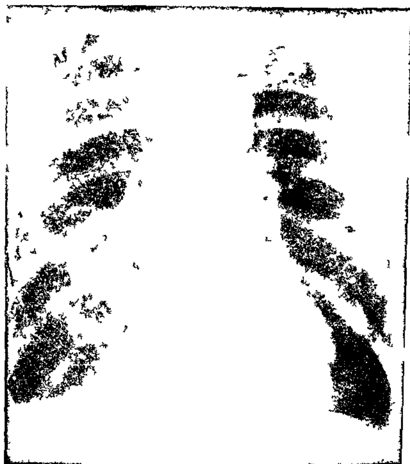


Fig 50 Unfolded Aorta
Associated with atheroma in an elderly man Note the prominence of

individuals of over fifty years of age The change is most marked distally near the iliac vessels and becomes less obvious towards the heart Atheroma causes some dilatation of the aorta sometimes localised but usually of a general nature Radiologically the

atheromatous aorta is somewhat widened and often very considerably elongated (Fig 50) This elongation causes the vessel to curve and bend in a characteristic manner Enlargement of the vessel results in less obstruction to the onflow of blood and it is clear therefore that atheroma does not add to the cardiac work Some rigidity of the vessel coexists, but neither does this change impose an added burden Advanced and widespread calcification of the aorta is often seen radiologically in old people who are free from cardiovascular symptoms Atheroma, however may attack the aortic valve cusps, distorting them to some extent and interfering with their proper closure Aortic regurgitation, with the typical murmur, may be produced in this way in elderly persons The amount of the leak is slight so that the blood pressure changes and pulsation of vessels characteristic of the lesion are not well developed An aortic aneurysm, more usually of the abdominal aorta may result from atheromatous disease But this does not affect the heart in any way, just as a backwater on a river has no obstructive effect upon the main stream It is upon the coronary arteries that atheroma produces its most serious effect, resulting in angina of effort, coronary infarction, and their consequences

Monckeberg's Sclerosis

This is a condition which involves arteries of the size of the radial and posterior tibial vessels and whose chief characteristic is the formation of calcified deposits in the medial coat of the arteries of the arms and legs These areas are at first distinct but in time they often amalgamate In the earlier stages the hard areas feel rather like the tracheal rings of a small animal and later the vessel as a whole feels continuously rigid This condition has no effect upon the heart or the blood pressure It is one of the common causes of peripheral failure of the arterial circulation manifesting itself for example by intermittent claudication, pallor, and eventually gangrene in the feet

CHAPTER XXVII

HYPERTENSIVE HEART DISEASE

THIS condition is essentially myocardial degeneration complicated by hyperpiesis or hypertension, or perhaps it would be more accurate to say that the hypertension is complicated by the heart failure, for the majority of such patients have already had a raised blood pressure for ten years or so without any cardiac symptoms or signs About 70 per cent of hypertensive patients die from heart disease Hypertension with no other obvious clinical cause is labelled "essential hypertension"

The cause of hypertension is still uncertain There appears to be good evidence that a degenerating area of renal tissue can produce a substance called "renin," which reacts with hypertensinogen to produce hypertensin Hypertensin is a substance which sends up the blood pressure, through the mechanism of the sympathetic nervous system Recent work on sympathectomy, in the course of which specimens of kidney tissue taken from living hypertensive patients have been found to be normal, suggests that renal degener-

cortex, caused by nervous or other stimuli, may be[*] sufficiently severe to produce renin, and thus hypertension, in the absence of organic renal changes Hypertensin causes spasm of the plain muscle of the middle coat of the arteries and arterioles Persistent spasm of this plain muscle over a long period of time causes hypertrophy of the arterial muscle coat When the hypertrophy has progressed to a certain point, or has persisted sufficiently long, the plain muscle becomes degenerate and fibrosis in it begins

It is well to consider separately the effects of these changes upon the cardiovascular system The blood pressure, of course, is raised during the first stage of spasm, but the increase is largely limited to the systolic pressure only At this stage the pressure can frequently be restored temporarily to normal by sedatives, rest in bed, starvation, purgation, and such measures, but a renewed increase soon follows

* *Studies of the Renal Circulation*, by J Trueta, A E Barclay, P M Daniel, K J Franklin and M L Prichard Blackwell Scientific Publications, Oxford, 1947

the cessation of treatment Later, the systolic pressure rises further and this increase is more persistent The diastolic pressure also becomes raised These changes impose an added burden upon the left ventricle, and all cases of essential hypertension show hypertrophy of this chamber The strengthened ventricle may support the added pressure successfully for many years so that all the functions of normal life are performed without symptoms At this stage the abnormality is often discovered, to the patient's surprise, in the course of some routine medical examination, as for life insurance

The raised pressure in the aorta produces a more efficient flow of arterial blood to the coronaries and to the heart muscle itself, and to this extent and in this particular it is beneficial Medial sclerosis is not usually found extensively in the coronary arterioles, and its consequences are compensated for by the improved coronary blood flow Thus the raised blood pressure, as such, and by itself, produces no deleterious effect on the heart

Most cases of hypertensive heart disease occur between the ages of 50 and 65, although a fair number of patients are between the ages of 40 and 50 There seems to be some racial distribution, for it is rare among the Chinese In some families there seems to be an abnormally high incidence of arterial disease Diet has no effect on hypertension, and the story of 'red meat' would appear to be a fable Hard physical work is not so important a causative factor as a state of sustained nervous tension in a patient's work Some patients date the onset of the trouble from the menopause, and prolonged thyrotoxicosis seems to accelerate the development of the condition The symptoms of hypertensive heart disease differ from those of myocardial degeneration without hypertension in being usually more acute and more severe Anginal pain is common and violent attacks of spasmodic angina are more frequent in the presence of hypertension Shortness of breath is usually the earliest manifestation of failure, and when failure intervenes in hypertensive heart disease the progress of the illness is generally rapid

Signs

The blood pressure may be persistently raised for many years without causing much enlargement of the left ventricle, so long as the coronary circulation remains normal With the advent of coronary atheroma the enlargement becomes obvious and rapidly increasing, and is due both to severe dilatation and to hypertrophy The aorta may not show much enlargement at first, but, as the condition becomes more chronic, it is more visible on the X-ray

screen both to the right and to the left of the spinal column (Fig 51) It is interesting that the enlargement is not so great post mortem as in life and this suggests that it is largely due to a stretching of the elastic tissue which subsides after death

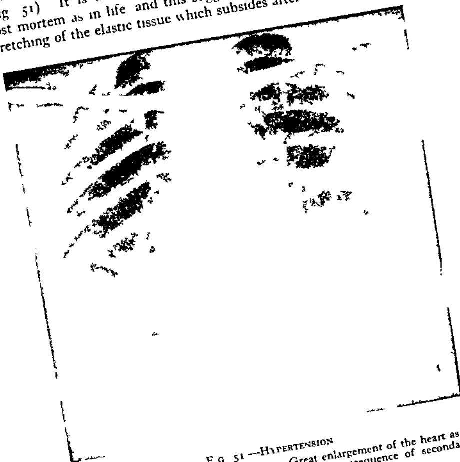


FIG 51 —HYPERTENSION

Transverse cardiac diameter 16 0 cm Great enlargement of the heart as a whole especially of the left ventricle This is a consequence of secondary coronary ischæmia from atheroma

The apex beat is displaced considerably to the left sometimes into the sixth space in rare cases even into the seventh It is forcible diffuse and sometimes there is a double thrust to it Arterial pulsation may be felt in the suprasternal fossa or lateral to this behind the right sternomastoid This is due to the elongation of the aorta and to the high position in the neck taken by the raised innominate and internal carotid arteries On auscultation at the apex the first sound is loud frequently impure and reduplicated When

the heart is under stress a third heart sound is *common, producing a* gallop rhythm The first part of this reduplicated sound has been thought to be due to auricular systole propelling blood into the left ventricle, the physical state of which has lost some of its normal laxity A systolic murmur is often heard at the apex, especially when the heart gets large The aortic second sound is loud and often

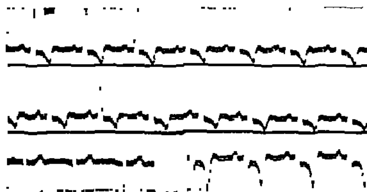


FIG 52—TYPICAL CURVE OF LEFT VENTRICULAR STRESS

ringing in quality The blood pressure is of course increased, an ordinary figure being 220/140 When the blood pressure is taken it may be noticed that pulsus alternans is present, the systolic pressure of the alternate beats varying, for instance, between the figures of 240 and 220 This is a sign of serious consequence, and usually indicates that life is not likely to extend beyond two years or so Aortic incompetence may supervene, from atheroma of the aortic cusps or stretching of the aortic ring

The electrocardiogram (Fig 52) in hypertensive heart disease always shows left axis deviation, and in a severe case frequently shows also inversion of the T wave in Lead 1, due to what is called "left ventricular stress" There may have been no shortness of breath and no coronary symptoms or signs in patients showing this change The T wave in Leads 2 and CR 4 is also sometimes inverted

Differential Diagnosis

A raised blood pressure may be found in conditions other than hypertension. Many individuals with a reactive visceral nervous system, especially young adults, are found, on routine examination, to have a greatly raised systolic blood pressure. A figure of 180 is common, and one of 200 not very uncommon in very nervous individuals. The notable fact, however, is that in such patients the diastolic figure is never raised to the extent seen in patients with true hypertension: thus, comparable figures in the normal cases above quoted would be 180/90, and 200/98. They nearly always have a nervous tachycardia, and their general reaction during examination suggests a considerable degree of suppressed excitement. The heart is of normal size and shape. The cardiogram is normal.

A much raised blood pressure is also found in cases of chronic nephritis. Here there is frequently a history of scarlet fever with an unusually long course, or even of scarlet fever without this suggestion of a complication, or possibly one or more attacks of severe tonsillitis. The urine is found to contain albumin, which is rarely present to any extent in hypertensive heart disease unless there is some myocardial failure. The urine in nephritis will show the presence of casts, epithelial cells, and possibly a few red blood cells. In congestive heart failure granular casts may occasionally be found, but never epithelial casts. The œdema in nephritis differs from that of heart failure in that it usually occurs without symptoms of failure such as shortness of breath, and in that it affects the loosely knit tissues of the body such as the eyelids and scrotum, equally with the feet and legs. In cases of doubt, renal function tests are useful, and the presence of an advanced retinitis in a young individual especially with papillœdema suggests primary renal disease. A final point is that a secondary anæmia of a severe degree is often found in patients with chronic nephritis, and is not a characteristic of hypertension. Coarctation of the aorta is a rare cause of hypertension in young individuals in whom there is no evidence of active disease.

In considering the stage of hypertension present, attention must be focused upon the relative proportions of arterial spasm and of chronic arterial change in the particular case. During the stage of spasm, variations in the blood pressure caused by rest, exercise, temperature, and emotion are considerable. When the stage of permanent change is well developed such temporary excursions from the fixed level are smaller and less easily produced.

Treatment of Hypertensive Heart Disease

The treatment of heart failure in hypertension differs in no important respect from the treatment of heart failure due to other causes One point should be stressed The administration of mercurial diuretics to a hypertensive patient who has in addition considerable disease of the kidneys may sometimes produce fatal anuria and uræmia The various complications present in the heart affected by arteriosclerosis such as auricular fibrillation auricular flutter, or cardiac pain of anginal type are treated exactly as though the raised blood pressure were not present

It is clear that the treatment of hypertension as such will be the most important part of the treatment of heart disease occurring in a patient with this trouble The treatment of hypertension has for generations been extremely unsatisfactory and since the condition is so common unnumerable drugs measures and regimens have been tried Hypertension like most diseases which are common and which fail to respond to treatment has been the happy hunting ground of multifarious quacks There are however certain therapeutic lines which are helpful in chosen cases

Adipose patients with hypertension occasionally succeed in lowering their blood pressure to a considerable extent and for a considerable time by reducing their weight This can be done satisfactorily only by strict dieting in which carbohydrates and fats are severely reduced In order to make up for the vitamin contained in the prohibited potatoes it is wise to prescribe ascorbic acid 50 mg once or twice daily in these cases Thyroid extract in small doses does not do harm and may help the reduction of weight Some patients with hypertension seem to date their troubles from *the menopause* and are occasionally helped by stilbœstrol and other appropriate treatment of this phase of life Patients with severe hypochromic *anæmia* are sometimes found to have a distinctly raised blood pressure which falls to a lower level and remains down when the microcytic anæmia has been adequately treated

There is no doubt that both *mental and physical activity* have a very marked temporary effect in patients whose blood pressure is raised Business worries domestic troubles and minor irritations are very provocative of an increase in the blood pressure This is probably the reason for the success of sedatives in the treatment of the disease Moreover it is extremely important to remember that a raised blood pressure may be present for fifteen or twenty years in many cases without causing any serious bodily trouble This fact

should be impressed, whenever it is legitimate, upon patients who know that their blood pressure is raised The "blood pressure conscious" patient who is anxious to know the figures every time he is examined is a danger to himself This state of mind can frequently be helped enormously by an explanation of the chronicity and harmlessness of the disease in so many cases Physical rest also is extremely effective in lowering the blood pressure, so that patients admitted to hospital with a reading of 200/120 are often found to have a figure of, for instance 150/98 after four or five weeks rest This phenomenon is a windfall for the quacks, who stress the progressively falling blood pressure without revealing that the figure will return rapidly to the previous maximum as soon as the patient has been up and about for a few days

Sedatives The only type of drug which seems to have any definite and prolonged effect on the blood pressure is the sedative Phenobarbitone is most usually employed for its action lasts for some hours and it is relatively harmless The reality of the effect of sedatives is proved by the striking response of the blood pressure in suitable patients with hypertension to the doses of sodium amytal used to estimate the relative degree of spasm and of permanent arteriosclerosis before operative treatment of the condition

Diet For many years much has been taught and accepted with regard to the effect of diet upon the blood pressure The only relationship between red meat and red wine to raised blood pressure is that of sympathetic magic, for the patient s face is frequently plethoric and red also There is no real evidence to suggest that the type of food has a measurable influence upon either the symptoms or the progress of the disease It is true, however, that starvation lowers the blood pressure and it would seem reasonable to advise these patients to eat as little as keeps them comfortable

Drugs have been most disappointing as is seen by the list of remedies that have been tried for the condition throughout the ages Nitrites certainly lower the pressure, but their action is so fleeting as to be valueless Iodides have been used presumably on the assumption that they are supposed to dissolve fibrous tissue This is a relic of their known action in syphilitic gummatous lesions and has no basis in truth in any other condition Mistletoe and chloro phyll have been suggested both by legitimate practitioners and under fancy names by hard faced characters in advertisements but except that the remedies are obtained from romantic members of the vegetable kingdom they have no particular virtue Potassium thiocyanate has been used of late years and is perhaps the most

satisfactory drug hitherto employed It is also frequently disappointing, but may in some cases remedy symptoms even although the blood-pressure readings may not show much reduction The usual scheme of its administration is as follows during the first week 0 1 gramme of potassium thiocyanate in ½ oz of chloroform water is given t d s, p c Provided the serum concentration of the drug remains between 5 and 10 milligrammes per 100 cubic centimetres this dose is then continued twice daily If the thiocyanate concentration in the blood is more than 10 mg per cent then ten doses of 0 1 gramme are given during a period of a week, in doses arranged so that some is administered each day If the serum concentration falls below 4 mg per cent, 0 1 gramme is given four times daily for the next week The dosage must be regulated by careful estimation of the blood concentration of potassium thiocyanate which must be done at first at intervals of a week The figure should be kept at between 5 mg per cent and 10 mg per cent When the blood concentration of the drug remains constant the blood thiocyanate need be estimated only at such intervals as once a fortnight once a month, and then finally once every two or three months

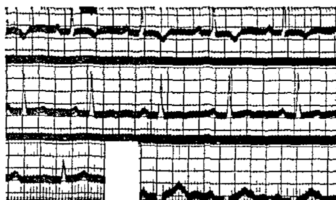
Venesection This measure is frequently symptomatically beneficial, but does not very often succeed in reducing the blood-pressure figures appreciably or for long

Operative Treatment Two varieties of surgical treatment have been tried for hypertension On the analogy that a degenerating area of renal tissue is a source of renin removal of one severely diseased kidney has been frequently tried, and occasionally with success, so that in such a case the blood pressure falls dramatically and for a long period of time But on the whole these cases are very rare, and the the success of the operation is problematical

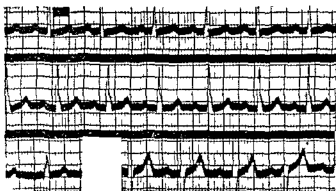
The other surgical measure is sympathectomy For some years this has been tried, either in the form of supradiaphragmatic sympathectomy, or as lumbar sympathectomy Both of these measures have had a variable success, but Smithwick's operation, combining both measures, seems to have made the success of the procedure more definite and more permanent Smithwick's operation involves removing or cutting the following structures on both sides of the body the great splanchnic nerve, which is a pathway of the sympathetic thoracic roots D 6 to D 9, the lesser and least splanchnic nerves, D 10 to D 12, and the lumbar sympathetic roots, L 1 and L 2

The effect of this operation is to produce a very widespread vasodilatation over a large area of the body, so that perforce the blood pressure must fall Hyperæmia of the renal cortex is also a

result of sympathectomy Since cortical ischæmia can be causec by experimental stimulation of peripheral nerves in animals it i



(A)



(B)

Fig 53

with hæmorrhages and exudate simultaneously disappeared There is an interval of eleven days between the date of the final operation and the date of the second tracing

reasonable to regard sympathectomy as probably beneficial in this way also A renal cortex rendered ischæmic by spasm could produce renin in the absence of permanent organic disease It is interesting that the postoperative fall in addition to the early rapid change

frequently continues over a subsequent period of up to twelve months Cases have now been followed up by White and Smithwick for as long as seven years and their experience comprises something like one thousand cases in all (*Personal communication*) It would seem that the effect produced by this form of sympathectomy is in a sufficient proportion of cases of long duration The operation is of course not without risk

The effect upon the heart is parallel to that upon the blood pressure so that symptoms of heart failure disappear the enlarged heart shrinks in size and the electrocardiogram of left ventricular stress reverts to normal in some cases (Fig 53)

The treatment is not one to be lightly undertaken and cases for it must be chosen with the utmost care Patients between the ages of 30 and 40 years react more satisfactorily than those of between 40 and 50 and in those over 50 the risk is considerably increased Women seem to be better subjects than men A small pulse pressure —for instance a figure of 190/136—provides a better outlook than would a pressure of 190/100 One explanation may be that in established arterial disease with much vessel rigidity the diastolic pressure tends to fall Many untreated individuals with hypertension maintain a fairly high but steady blood pressure for years without symptoms If there is in addition a family history of prolonged and relatively harmless hypertension it is wise not to embark upon sympathectomy in such cases without full and good reasons If however the high pressure is beginning to rise definitely and progressi [illegible] be clearl[illegible] threatened b[illegible] considered [illegible] blood pressure retinitis with papilloedema [illegible] insufficiency often in a young adult is no absolute contraindication to this measure

It is essential to discover in patients who are candidates for sympathectomy that no severe degree of renal disease is responsible for the hypertension and further that there is no evidence of renal failure as judged by urea clearance tests and other similar measures Serious myocardial degeneration is also a contraindication to operation Certain tests will reveal the amount of spasm in such patients and the outlook is better in proportion to the degree of spasm The cold test is useful and indicates the extent above the normal resting level to which vascular spasm can force up the blood pressure The blood pressure is first taken at intervals of a minute with the patient lying at rest The forearm is then immersed in ice water immediately

after such a blood-pressure determination The blood pressure in the opposite arm is recorded half a minute after the immersion and again after the second half-minute at the moment when the forearm is withdrawn from the iced water The blood pressure is subsequently taken at one-minute intervals until it returns to the previous resting level The administration of sodium amytal indicates the extent to which the arterioles can be relaxed from the normal resting level Three doses of sodium amytal (3 grains per dose) at intervals of one hour are administered to the patient, who has been resting for a day or two in bed The resting blood-pressure level before the first dose is determined and the blood pressure is then taken at hourly, or if necessary half-hourly intervals In satisfactory cases the pressure is seen to fall, for example from 190/120 to 140/90 The speed of the fall is an important factor, as well as its extent

CHAPTER XXVIII

CARDIAC INFARCTION OR CORONARY THROMBOSIS

A CLOT is not always found post mortem in patients who have obviously died as a result of cardiac infarction although other myocardial and pericardial changes may be typical In such cases it is possible that a sufficient state of local ischæmia is capable of producing the infarct without previous obstruction of the nutrient vessel by an organised thrombus It is for this reason that cardiac infarction is the better descriptive term

However, in most cases a cardiac infarct is caused by coronary thrombosis Such thrombosis does not occur in a healthy vessel The underlying lesion is atheromatous in type This atheroma may be either early or well developed Although the left descending interventricular branch is the commonest site for coronary infarction this lesion may involve any other branch Owing to the anatomical variability of the coronary arteries infarcts are usually called anterior or posterior, rather than left or right coronary Syphilis only nips the coronary mouths by œdema or fibrosis from an aortitis, and therefore does not cause coronary infarction When this complication attacks syphilitic cases an associated atheroma is always the nidus of deposition for the coronary clot

Two other rare conditions may also cause coronary infarction embolism and periarteritis nodosa Coronary embolism is a rare complication of infective endocarditis, for in most of such cases the abruptness of the vascular obstruction causes sudden death But occasionally the patient may survive and the signs are then those of a cardiac infarct Periarteritis nodosa, in cases where the nodular inflammatory lesions occur in the coronaries, may show multiple infarction

The immediate pathological results of an infarct may be seen in the pericardium, the myocardium, the endocardium, and the blood The later results may affect directly or indirectly other organs in various ways

The infarct usually extends to the *pericardial* surface, and local
peric
may
have

The *myocardial* area which has been infarcted partly recovers as the collateral circulation develops, and partly necroses If the necrosed area is large, rupture of the heart may occur Cardiac infarction is the chief cause of rupture of the heart If the necrosed area is of moderate size, fibrosis replaces it Here again, depending upon the local blood supply, a weak area may be formed which becomes a cardiac aneurysm, or a firmer fibrotic area may result, the muscle being correspondingly weakened, or a small fibrous scar only is left which produces no deleterious result Cardiac aneurysms in patients who survive often eventually show some calcification, and this fact helps in their radiological diagnosis The reaction of the remaining normal heart muscle depends upon the state of the rest of the coronary circulation If this is good, and the infarct has not caused too much myocardial destruction, a perfect functional recovery is possible by hypertrophy of the remaining heart muscle At the other end of the scale is the patient in whom, as a result of persisting coronary ischæmia in other areas, or because of the extent of the myocardial destruction, congestive heart failure quickly supervenes Most cases lie between the extremes of complete recovery and a progressive cardiac deterioration In this middle group the myocardium will recover as compensatory hypertrophy occurs but the recovery will be to a lower functional level than before Such recovery is often permanent at the new level, and provided the physical demands can be adjusted to this, the state of the heart on balance will remain adequate Should the coronary disease be widespread, further infarctions may follow Here the subsequent pathological pictures are reproductions or accentuations of the first

The *endocardium* over the infarct is often inflamed, and clot may be deposited on it This sometimes gives rise to a complication; embolism of the brain, or of some other systemic organ, may follow when a portion of this clot becomes detached from the left ventricle Similarly, pulmonary embolism may follow infarction of the right ventricle This embolic complication usually follows the infarct after an interval of one to three weeks The infarcted muscle itself has an irritative effect in some cases, for ventricular premature beats or even ventricular paroxysmal tachycardia may be set up, and, occasionally, ventricular fibrillation and sudden death may result The septum may be infarcted, and this may result in the production of intraventricular block, the conducting tissues being involved Rupture of the septum has been described The signs of this are similar to those found in congenital interventricular septal defect *

Fever leucocytosis and an increased erythrocyte sedimentation rate are common general bodily reactions to coronary infarction during the acute stage

Ætiology

Most cases of coronary infarction occur between the ages of 50 and 60 years, but the disease is common between 60 and 70 It occurs between 30 and 40 and rarely is seen in the second decade It is considerably more frequent in men The pain and prostration are more severe in sensitive and overworked professional men and women Some families appear to be predisposed to the condition

Symptoms

The symptoms are due to myocardial ischæmia which produces pain shock and impaired cardiac function

The severity of the pain varies according to the sensitivity of the individual nervous system In some it is agonising in many severe in many only moderate in some slight and in a few absent Attention must therefore be fixed for diagnostic purposes upon its other characteristics In position it is usually central either retrosternal or even epigastric From this position it may spread on either side laterally for a few inches or to the arm or arms It may radiate through to the back or up to the neck Radiation is less in extent than is seen in angina of effort During a period of a week or so before the infarction becomes established one or more attacks of pain slight in degree but over the typical area may have been noticed but in many cases no such warning pains are felt The two chief diagnostic features are that the pain is rapidly maximal and slowly waning and that it has a tendency to appear first at night or at rest

A typical history would be as follows a patient who has never before suffered from cardiac pain or a patient who has suffered from classical angina of effort is suddenly seized while at rest with a severe central retrosternal pain which rapidly becomes agonising It waxes and wanes somewhat but does not subside completely until therapeutic injection of morphia some hours later It then returns and morphia may again be required It slowly subsides during the next day or more The total duration is often four or five days

A second history might be of a similar onset of pain rather less severe and unrelated to exertion which after lasting for an hour or so spontaneously ceased only to recur and again subside several times, such recurrences being spread over several days and the later attacks being milder

A third history is one that simulates angina of effort Here, in a patient previously healthy, pain of angina of effort type begins suddenly and severely, ceasing with rest But as days go by the severity of the pain becomes less, until after the lapse of weeks perhaps it is no longer felt

Sometimes the pain is relatively mild and its total duration as brief as an hour or two Here the patient may remain up and about as usual, although feeling below par Silent infarcts occasionally occur

The adjacent pleura is in some cases inflamed, causing pleural pain on breathing in addition to the cardiac pain Impairment of the cardiac function causes dyspnœa In large infarctions this is present at rest, but in smaller ones is absent unless exercise is taken In ambulatory cases it is nearly always to some extent noticed Shock is frequently profound, and is the chief cause of the great fall of blood pressure Pallor, sweating, and tachycardia are its chief manifestations The shock is not necessarily proportional to the size of the infarct Exhaustion is often great and in nearly every case lassitude is a prominent complaint Flatulence, vomiting and radiation of the pain to the abdomen may suggest an abdominal rather than a cardiac lesion

Signs

The immediate physical signs of the infarction are pallor, often associated with a slight cyanosis, an anxious look, and sweating The respirations are increased in rate Fever is present, but, owing to shock, the mouth and axillary temperatures may not reveal it Leucocytosis and a raised sedimentation rate are usual Some orthopnœa is noticeable The pulse is of poor volume, and the rate rapid The neck veins may be abnormally full The apex beat, if palpable, is usually displaced to the left, and is often flabby The heart sounds are distant, the first often being of poor quality or reduplicated A good-quality loud first sound is rare in cardiac infarction A systolic apical murmur may be present The rhythm may be disturbed by premature beats, even in the presence of tachycardia In rare cases auricular fibrillation or paroxysmal tachycardia may have supervened Pericardial friction, usually distant and soft, may be audible

The blood pressure is often greatly lowered, largely from shock and partly from lowered myocardial efficiency, but this reduction is in some cases slight so that unless previous figures are available it is not noteworthy Where the infarction is small the blood pressure may not be affected If it is possible to examine the

pulmonary bases fine râles are often audibl I I urinary output may be temporarily reduced from the preser t sh ck

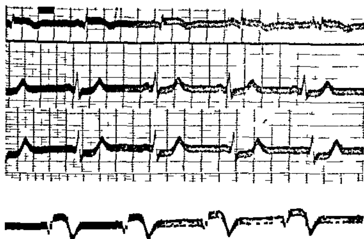


FIG 54 —ACUTE ANTERIOR CORONARY INFARCT

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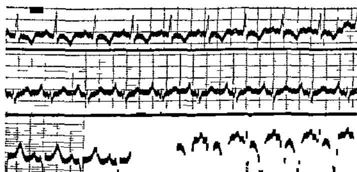


FIG 55 —ACUTE ANTERIOR CORONARY INFARCTION—SEVEN MONTHS PREVIOUSLY

n Le ds I and As does ession

The electrocardiogram (Figs 54 to 57) is generally diagnostic although even here the rule is not absolute for in some cases of

posterior infarction no change is observed in the four routine leads Generally within two or three hours of the onset the S-T part of the

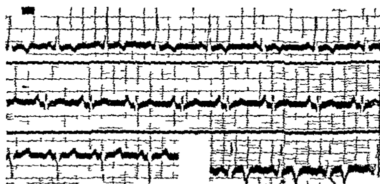


Fig 56

d sappeared



Fig 57—Anterior Infarction

2 and 3 are a t p cal S-T had a typ cal

tracing is elevated in Lead 1 and depressed in Lead 3 if the infarction is anterior and the reverse changes are seen if the lesion is posterior

During the first week after an anterior infarction the S-T deviation lessens, and the T wave in Leads 1 and CR 4 tends to become sharply depressed, diphasic, or inverted. The inversion is commonly sharply pointed. Later the T wave may slowly return to its previous shape, or it may be flattened, or may remain inverted. The R wave

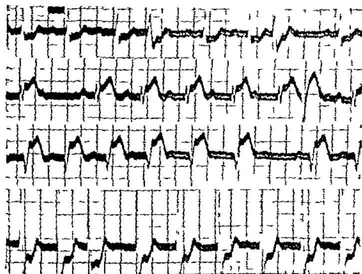


FIG 58.—ACUTE POSTERIOR CORONARY INFARCT

complex in Lead 2. A premature ventricular beat is visible at the end of Lead 2 and others after the third and fifth normal ventricular complexes in Lead 1

in anterior infarction, becomes absent or barely present in Lead CR 4, and its absence is often the only remaining sign of previous infarction.

In posterior infarction the S-T complex is elevated in Lead 2 and in Lead 3, but this elevation slowly lessens and disappears (Figs 58 to 60). A corresponding S-T depression is usual in Lead CR 4. The T wave in Leads 1 and CR 4 remains abnormally prominent, but that of Lead 3 soon becomes inverted, and may remain so. The Q wave in Lead 3 soon becomes abnormally large.

Finally it may be affirmed as a brief generalisation that any abrupt

abnormality which occurs in the shape of the QRST part of the electrocardiogram and which then shows a slow return to normal is probably due to coronary infarction This is particularly so if the

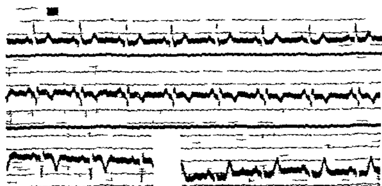


FIG 59—POSTERIOR CORONARY INFARCT—THREE WEEKS AFTER THE ORIGINAL ATTACK

There is still slight S T elevation in Leads 2 and 3 with sharp inversion of the T waves in Leads 2 and 3 Another characteristic of the curve of patients who have suffered a posterior infarct is the deep Q wave present in Leads 2 and 3 which is well shown This deep Q wave is more prominent in Lead 3 than in Lead 2 and often may be seen only in Lead 3

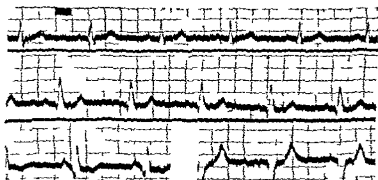


FIG 60—POSTERIOR CORONARY INFARCT—ONE YEAR PREVIOUSLY

The only abnormality of the T wave is the inversion of it in Lead 3 The Q wave is clearly deep and prominent in Lead 3 and is deeper in Lead 3 than in Lead 1 or CR 4

patient has suffered from an attack of cardiac pain at the time of onset Acute pericarditis and pulmonary embolism are much rarer causes of somewhat similar electrocardiographic changes associated with pain Here also T wave inversion and S–T deviation occur

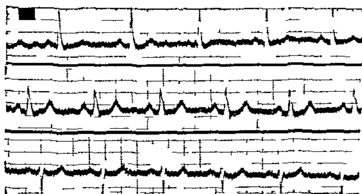
Treatment

The patient should rest in bed in the p t n m t comf rtable to himself A heart bed is s metimes ad able f rthopn ea s



F G 61

F g 61 is from a pat ent ho had he t p cal h ory of a co onary infa ct two mon hs pre ously It shows a defin e left bund e branch les on of the characteri t c type



F G 62

F g 62 shows a trac ng f om the same pat ent six mon hs later The e

marked Unnecessary propping up or uncomfortable flatness of posture sl ould both be avo ded

Pain, shock, and anoxæmia all require individual attention in the acute stage. Morphine should never be withheld if the severity of the pain demands it. Doses of ¼, ⅓, or even of ½ grain are given subcutaneously, and may be repeated if necessary at intervals of four hours. Some patients are susceptible to morphine, and vomiting may become distressing. In them diamorphine (heroin) may be substituted. Pethidine, 50 to 100 mg. by mouth, or 50 mg. intravenously, is valuable in some cases.

Shock should be treated by warmth, and by the injection of such a drug as *anacardone (nikethamide)*. *The action of these is on the* medullary centres. Doses are usually 0.5 to 1.25 grammes in 1.8 c.c. every four hours intramuscularly, for 2 or 3 doses. If shock is profound, and fails to respond to treatment, adrenalin in doses of ½ to 1 c.c. of 1 in 1,000 solution may be given subcutaneously. In similar circumstances methedrine by injection can be tried, the dose being 15–30 milligrammes in 1.5 c.c. intramuscularly and 15–20 milligrammes in 1.5 c.c. intravenously for 2 or 3 doses at four-hourly intervals. Adrenalin is risky, as ventricular fibrillation and sudden death may result, but the severity of the shock may justify the risk.

Oxygen, by tent, mask, or twin nasal catheters, is often extremely helpful, especially in cases where pulmonary œdema or congestion is present. If ventricular paroxysmal tachycardia occurs quinidine *sulphate grains 3 to grains 5 at hourly intervals by mouth should* be tried. The maximum total dose here should not excede 40 grains in 24 hours.

The patient must rest in bed until the lesion is sufficiently healed, or until any associated heart failure has disappeared. Healing of the infarct is the prime consideration. Congestive failure if it follows, usually does so after an interval of time, during which, although dyspnœa and fatigue are present, a modified activity is still possible. The period of rest in bed generally advised is six weeks, but the duration is less in cases where the infarct is judged to be a small one. In severe cases the rest is absolute for a month or six weeks, but in small infarcts, particularly in old persons in whom a long period of recumbency may be harmful, absolute rest even in the *early stages may be inadvisable. The use of a commode in such* patients is often less exhausting than a bed pan. Moreover old people easily get hypostatic pulmonary congestion. The same relaxation is sometimes advisable in the very adipose.

Sedatives, such as phenobarbitone grain ½, can be given three or four times a day, provided the mental state remains quite clear. Theobromine sodium salicylate grains 5 to 7½ and theophylline

ethylene diamine in a dose of 1 to 2½ grains t d s p c, have also been given by mouth, but their beneficial effect is problematical The diet in severely ill patients and acute cases can be limited for a few days to well sweetened fruit juices, tea, or coffee Alcohol in moderation is not contraindicated After this period the patient is allowed solid, easily digested foods, a slop diet being avoided The bowels may be ignored during the period of shock and can then be moved on alternate days by enemas After seven to ten days the patient's customary aperient is allowed if necessary Sleep can be obtained by the use of the usual barbiturate drugs

Auricular fibrillation, paroxysmal tachycardia angina of effort, and congestive heart failure may follow coronary infarction The treatment appropriate to the condition is given in each case

Prognosis

The immediate prognosis depends upon the severity and persistence of shock, and upon the size of the infarcted area The severity of shock can be measured by the degree and the persistence of the fall in the blood pressure, the tachycardia, the pallor, and the sweating It is harder to be sure of the size of the infarct Points suggesting a large infarct are continuance of the pain for more than four or five days, severity of the pain, marked dyspnœa, obvious distension of the neck veins, pulmonary œdema, considerable fever and leucocytosis, the presence of pericardial friction over a large area and for several days, the presence of a bundle-branch lesion, or of auricular or ventricular ectopic tachycardia, and marked electrocardiographic changes In a small infarct these abnormalities are absent Most patients recover from the attack If death occurs in the acute stage this is usually within the first two weeks After this the immediate danger is over Subsequent prognosis is concerned with the probable permanent effects of the infarct, and the possibility of recurrence When the patient is ambulatory cardioscopy is essential Prognosis is largely influenced by the size of the heart on measurement A small heart will suggest a good outlook, a large heart a bad one

A small infarct may heal completely and leave the heart as efficient as it was previously The patient's occupation must be taken into account *Complete symptomatic cure is less likely in a heavy* labourer than in a more sedentary or a brain worker Recovery to 70 or 80 per cent of cardiac efficiency is common, and if the cardiac requirements can be proportionately reduced the equivalent of a complete cure is achieved Permanence of such cures is good,

the heart continuing to function well for many years. The electrocardiogram in these patients often returns to a normal shape.

In some cases the symptoms and signs of congestive heart failure with œdema follow the infarct. Although as a rule this condition is progressive, certain individuals may recover from such a state so satisfactorily as to lose all evidence of congestive failure. Such patients may return to a normal life and may keep well for a matter of years although engaged on quite hard physical and mental work. In other cases the myocardium is so damaged as to be incapable of much exertion, and dyspnœa, lassitude, and fatigue are prominent. Here maintenance of efficiency even at a reduced level is less probable. Such cases usually show enlargement of the heart and abnormal electrocardiograms. In all types of case unexpected death may occur, spontaneously or after some sudden physical effort.

In view of the excellent chance of a complete functional recovery, undue gloom must be avoided at all costs, the patient being encouraged to add slowly to his physical exertion. Provided that this addition is gradual, it is safe to allow such a patient to return to a full physical life, entailing moderate and not excessive effort. He should be warned to avoid undue dyspnœa or undue exertion. One patient, after a serious infarction associated with the onset of auricular fibrillation which lasted for five weeks, subsequently underwent a partial thyroidectomy for Graves's disease, and after convalescence returned to his hobby of cutting and splitting timber. He was still well and at work nine years later. If an optimistic and carefully constructive view is not taken, extreme care and undue caution being substituted, there is grave danger that many patients may drag on for years an unnecessarily passive existence, as useless and unhappy cardiac neurotics. After an infarct, as many patients probably become victims of medical pessimism as suffer physically from the results of the disease. The probability of a second attack is about four to one against.

Differential Diagnosis

Other conditions giving as severe pain are gall-stone colic, acute pancreatitis, perforation of an abdominal viscus, dissecting aneurysm, and spasmodic angina. *Gall stones do not cause cyanosis or dyspnœa*, but coronary infarction may cause a sallow almost yellow appearance, and the pain is sometimes chiefly abdominal. Between attacks of gall-stone colic the patient remains well. *Perforation of a viscus* is associated with a minimal respiratory movement, with maximal abdominal rigidity, whereas in coronary infarction there is usually

some dyspnœa or slight hyperpnœa, and any associated upper abdominal muscular spasm can be overcome by gentle persistent pressure In most cases of *dissecting aneurysm* the pain starts with great abruptness and is referred to a lower level of the body, and shock is often extremely severe The electrocardiograph may provide the only means of differential diagnosis for in dissecting aneurysm there is typically no change in the electrocardiogram

Pulmonary embolism, with sudden stress upon the right side of the heart, may simulate coronary infarction closely Extreme dyspnœa and tachycardia, hæmoptysis and pleural friction, or localised râles, all suggest embolism The electrocardiogram is often normal but may be misleading, for inversion of the T wave in Leads 2 and 3 and a deep Q wave in Lead 3 are found in both pulmonary embolism and in posterior infarction The right pectoral lead however (CR 1) shows inversion of the T wave in pulmonary embolism and an upright T wave in posterior infarction Pleurisy, pneumonia, and pneumothorax all occasionally cause difficulty in differential diagnosis

Coronary infarction may masquerade as angina of effort Spasmodic angina may give rise to attacks of very severe pain But between these attacks effort pain and dyspnœa are not increased, and there is no obvious deterioration in the patient's condition such as would occur after a series of infarcts following one another closely

CHAPTER XXIX

ANGINA OF EFFORT

THE pain of angina of effort is the most characteristic symptom of coronary disease It is an indication that some portion of the ventricular muscle is being made ischæmic by exercise The two lesions which most commonly interfere with normal coronary flow are atheroma of the coronary arteries, and syphilitic aortitis Atheroma is usually found within an inch or two of the coronary mouths, and syphilitic aortitis nips the coronary vessels as they pass through the swollen or fibrotic aorta The atheromatous lesion may cause a loss of elasticity only, so that the blood flow is fully adequate at rest, or during slight exercise, but when greater effort is made this local loss of elasticity does not allow as full a coronary vasodilatation as is desirable In other cases the vessel has a normal but rigid lumen, and in others still a definite narrowing is present Pain does not invariably follow coronary narrowing Cases are found in which no pain existed during life, although a post-mortem examination shows that coronary atheroma had caused myocardial degeneration The sensitivity of the nervous system is an important factor determining whether pain is present or absent, severe or slight

Angina of effort is sometimes the only manifestation of a slight coronary infarction But in this case the pain is characterised by suddenness of onset, and by the fact that it is most severe at its first appearance, and that it diminishes progressively in the days or weeks following the onset Angina of effort is also found in aortic valvular disease In aortic stenosis there is frequently much local calcification of the valve, and similar atheromatous change sometimes has involved the coronary ostia also In aortic regurgitation the diastolic blood pressure and the mean pressure during diastole are lowered Since diastole is the period during which the heart muscle receives its blood supply, it is not surprising to find severe anginal pain occurring in some cases of advanced aortic incompetence Another factor in the production of angina of effort may be severe anæmia Pernicious anæmia is the type most frequently so concerned, but cases are met with in hypochromic anæmia, and rarely in the anæmia following severe gastric or duodenal hæmorrhage Here restoration

of the hæmoglobin percentage to normal nearly always removes the cardiac pain, often permanently There is probably a slight local loss of *coronary elasticity in such cases insufficient by itself to cause* symptoms

A difference in the blood supply to adjacent areas of heart muscle seems to be necessary for the production of angina of effort a *relatively ischæmic area being present amid a mass of normal muscle* If the whole ventricular muscle suffers from oxygen lack as in severe congestive failure due, for example to mitral disease no angina of effort is produced That coronary disease is the basic *cause of angina of effort becomes increasingly certain as fresh* evidence becomes available Of 112 such cases in hospital and private practice analysed by Dr R Bodley Scott and myself 88 per cent had clear evidence of vascular disease and of the remaining *12 per cent a further 2 per cent showed changes only in Lead CR 4* of the electrocardiogram

Both coronary disease and angina of effort are considerably commoner in men and in patients over fifty years of age A positive *family history of coronary disease is significant* although *in many* cases this is lacking One male patient of mine who suffered from angina of effort following coronary infarction had three brothers, two of these had died of coronary infarction and the third had [illegible]

[illegible] In

[illegible] n, in

[illegible] the rema n ng 12 per cent it was [illegible]

to the left arm, in some cases to both arms and rarely to the right arm only Sometimes it travels to the throat, the tongue, or the angle of the jaw It can be present in the arm only

The great diagnostic feature of angina of effort is that it is *quantitative*—that is that it corresponds exactly in intensity and in time of onset with the amount of exercise undertaken This amount differs greatly from case to case The more observant the patient the more accurately is the quantitative characteristic revealed A man who can walk at two miles an hour without pain begins to get it if the pace is increased Hills accelerate the speed of onset, as does cold weather It is often more easily brought on after meals, possibly because the accumulation of blood in the splanchnic area cuts down the supply to the heart and to the other systemic organs It may be worse in the morning than later in the day In some cases it is

particularly noticed after getting into bed, the cold bedroom or sheets or the exertion of undressing after climbing stairs may be factors here Some patients can walk off the pain, so that, although it pulls them up once or twice it then disappears Coronary spasm without definite narrowing must be concerned in these cases It is important to discover what amount of exertion such patients can undertake when at their best, for in this way it is possible to obtain a clue as to the functional powers and the pathological state of the ventricular muscle

The pain rarely lasts for longer than a few minutes, though in some hypersensitive individuals it may be said to continue for half an hour after an acute attack Nor is the pain in simple angina of effort usually severe At its commencement it is more an oppression in the upper chest, or a sense of tightness The patient stops for a rest before the pain becomes severe, in order to allow it to subside A sense of hurry or mental tension, as in a walk to catch a train, is an additional factor which predisposes to an attack of pain Emotion, whether pleasurable or painful, causes typical coronary pain in many of the more nervously sensitive cases Since an injection of adrenalin has the same action, it is reasonable to suppose that the mechanism here is one of coronary spasm

Spasmodic Angina

In a few cases this spasmodic factor is so great that it colours the whole clinical picture, and the writer labels these cases spasmodic angina " In them the amount of exertion necessary to cause pain is negligible, indeed a digestive upset, an unpleasant thought, or a bad dream can cause an attack of pain The pain of spasmodic angina, as described by patients who are good witnesses, is different from that of effort angina, from which they usually also suffer The spasmodic attack is fired off by some small stimulus, and then, in the words of one patient, it "has to run its course The pain in spasmodic angina is far more severe than that of effort angina, and it is in these rarer cases and in those of coronary infarction that a sense of impending death may occur This feeling is never found in classical angina of effort Pallor and sweating may be found in spasmodic angina and the blood-pressure reading is increased during the spasm Cases of aortic regurgitation with coronary pain are especially liable to angina in its spasmodic form

Angina of effort and spasmodic angina are both relieved by nitroglycerin and amyl nitrite This is an important diagnostic point distinguishing them from other types of chest pain

A curious point about angina of effort is that it disappears in cases where auricular fibrillation supervenes. Nor is this pain seen to develop in cases of auricular fibrillation. A fact worth impressing upon patients is that death does not occur in an attack of pain of the angina of effort type. In none of a series of 112 analysed cases did this happen.

Dyspnœa is an important symptom. Patients who get pain without dyspnœa have on the whole a healthier myocardium than patients whose primary symptom is dyspnœa and who on exertion also get some anginal pain. Orthopnœa is a fairly common associated symptom, and many patients who get slight spasmodic attacks at night find that a more upright position relieves them. This symptom suggests severe myocardial degeneration.

Signs

Physical examination reveals no characteristic signs, but the associated coronary lesion may or may not have been sufficient to cause myocardial degeneration and consequent cardiac enlargement. In a number of cases no signs of disease are discoverable clinically or by instrumental means. But in the majority one or more of the following clinical changes are found: cardiac enlargement particularly of the left ventricle, changes in the first apical sound, such as poorness of the quality or reduplication; murmurs, apical systolic from ventricular dilatation, basal systolic from atheroma of the aortic cusps, or aortic diastolic from aortic regurgitation, changes in the blood pressure, hypertension of the usual type, or in some cases a significantly raised diastolic with a normal or only slightly increased systolic pressure, cardiac irregularities of force or rhythm such as pulsus alternans, or premature beats usually ventricular in origin. One final important clinical point is that hyperæsthesia over the chest is uncommon in angina of effort, but it is a constant feature of non-organic functional cardiac pain.

In addition to these abnormal physical signs some important electrocardiographic changes are found in patients with angina of effort. Whether the changes indicate previous minimal and subclinical infarction is not clear. This may well be the case in the more definite abnormalities. The commonest of these changes are an enlarged Q wave in Lead 3, some flattening of the T waves in Leads 1 or 2 or in both, and inversion of the T waves in these Leads, S–T deviation of the T_1 or T_3 type, depending upon whether most of the coronary disease is anterior or posterior. In some cases the classical Leads 1, 2 and 3 are normal, but S–T deviation, diminished

Q wave, or M-shaped QRS complexes are visible in Lead CR 4 In other cases the electrocardiogram may be normal at rest, but may manifest one or more of the above changes after exercise Bundle-branch lesions and other abnormalities indicating widespread or advanced local ventricular disease may be present, but these are manifestations of the results of established myocardial degeneration, rather than manifestations of the partial coronary ischæmia, which is responsible for the coexistent state of angina of effort Auricular fibrillation and complete heart block are very rarely, if ever, associated with angina of effort Should coronary infarction occur as a complication of angina of effort, the changes typical of that condition are of course present

The cardiac enlargement, measured orthodiagraphically usually involves the left ventricle Aortic enlargement, usually due to atheroma but in a few cases to an associated syphilitic aortitis, is often seen

Prognosis is difficult in this condition The outlook, on the whole, is immeasurably better than is usually imagined The grim pictures of fatal attacks in past literature, lay and professional, are probably drawn from cases of coronary infarction Death does not occur in the attack of angina of effort A bad outlook is suggested by the following associated symptoms and signs severe dyspnœa on exertion orthopnœa, paroxysmal nocturnal dyspnœa or cardiac asthma, and marked cardiac enlargement The danger of intercurrent coronary infarction must be taken into account This occurs in about 25 per cent of cases In view of the uncertainty of the outlook, and of the unexpected survival for many years of some patients, it is wise to give as hopeful a prognosis as possible A near relative should be warned of the dangers of coronary infarction, and of unexpected death, but to make the patient unnecessarily apprehensive is injustifiable, and is likely to impede the best line of treatment, which is temperate and judicial reassurance, and slowly graduated exercise

Treatment

In slight or moderate cases the following measures are the most satisfactory A really complete cardiological examination is advisable, both to estimate the condition of the myocardium and to convince the patient that he can rely upon the advice subsequently given Rest in bed is usually unnecessary, except in prolonged and rather severe cases, when it is occasionally extremely helpful A period of two to four weeks is sufficient Regular walking exercise is encouraged It should be on the flat, and at a speed and for a

distance just below the pain level This it can be explained will keep the myocardium in good tone and will stimulate a collateral coronary circulation to develop which will take over some of the work of the atheromatous artery Golf on the flat and similar exercise within reason and below the pain level is good If a patient can walk off his pain he should do this *Hurry and stress or a feeling of drive or tension must be consciously avoided* Such an individual must be made aware of his keyed up nervous system and must learn to control it and relax *Laisse aller* is a good motto Rest should be taken immediately after meals for half an hour or so if possible

The diet varies with the case Individuals who are overweight will benefit from slimming but this should be done by diet alone for thyroid extract has a tendency to increase the acuteness of coronary pain Dyspepsia must be appropriately treated Patients with nocturnal attacks of spasmodic angina are often better with a small meal in the evening and in them gastric distension may be further relieved by a glass of hot whisky or a 60 minim dose of 'three spirits in hot water spirit of cajuput spirit of chloroform and aromatic spirit of ammonia in equal parts just before retiring Tobacco particularly in the form of cigars is contraindicated in anginal cases Strong tea and coffee may also cause dyspepsia and sleeplessness Diabetics not infrequently suffer from coronary disease and in them the carbohydrate content of the diet should be maintained at at least 150 grammes per diem if necessary by the help of insulin a figure of 200 grammes is more desirable The myocardium needs glycogen and a starvation diet will interfere with this need Alcohol in reason is not forbidden Certain physical therapeutic measures are useful in some cases Individuals with a pendulous protuberant lower abdomen compensate for this with a backward curve of the dorsal spine and a falling in of the upper thorax They are helped sometimes by an elastic lower abdominal belt, and by regular breathing exercises

The drug treatment of angina of effort is on the whole rather disappointing The pain can be controlled and temporarily prevented by glyceryl trinitrate or amyl nitrite and this action is of course of great value when the attacks of pain are severe or frequent Amyl nitrite by inhalation in doses of 3 to 5 minims is best reserved for severe attacks of spasmodic angina Angina of effort is most helped by glyceryl trinitrate in *doses of $\frac{1}{150}$ to $\frac{1}{60}$ of a grain* These tablets are made up with chocolate in order that they may be crushed and held for some minutes under the tongue or in the mouth If

they are swallowed whole their effect is less rapid and good The tablets may be taken without harm as often as is required during the day, either to lessen pain, or prophylactically just before the undertaking of some exercise likely to produce pain Erythrol tetranitrate, taken three times daily, is sometimes valuable Its more prolonged action prevents the onset of pain, when attacks are frequent and easily provoked The dose is ½ to 1 grain t d s , p c , but this can be increased gradually to 1½ or 2 grains t d s , p c Phenobarbitone ½ to 1 grain t d s , p c , is a useful general measure It should be omitted periodically for two to three weeks at a time, to prevent cumulation It may be given in tablets, or in a mixture with theobromine sodium salicylate 7½ to 10 grains The theobromine compounds are often given, but the effect in any given case is quite unforeseeable The most usual drugs are theobromine 5 to 7½ grains, theobromine sodium salicylate (diuretin) 10 to 15 grains, euphyllin (theophyllin ethylene diamine) 1½ to 3 grains, theobromine calcium salicylate 5 to 15 grains, and theobromine sodium acetate 5 to 15 grains t d s

Very severe and intractable angina of effort, with or without equally or more severe attacks of spasmodic angina, may need surgical treatment The chief first indication for surgical treatment is that the patient's pain is getting progressively worse, or continues to be very severe after full trial of other measures, so that life is unbearable or miserable There are three measures which have been applied in these cases sympathectomy, thyroidectomy, and omentopexy The first of these to be tried was sympathectomy The operations have varied in extent The least severe is section of the sympathetic trunk between the superior and middle left cervical ganglia A more complete operation comprises removal of the upper middle and inferior (Stellate) cervical ganglia These operations are not without risk, and Horner's syndrome usually follows them A slightly different operation has been introduced by Raney* which avoids the production of a Horner's syndrome It consists of paravertebral exposure of the sympathetic rami from behind, of section of the grey rami 2 to 5, and of trans-section of the sympathetic chain between the 5th and 6th ganglia The rationale of other sympathectomies has been the obliteration of afferent sensory impulses from the heart, Raney's theory postulates that in coronary disease relief from spasm rather than passive dilatation follows sympathectomy He therefore claims that his operation prevents exercise, emotion, or other causes from producing coronary spasm

* R B Raney, *Journ. Am Med Assoc*, 1939, cxiii, 1619

Thyroidectomy to be effective must be complete If the cases are properly chosen the results are good The operative risk is very slight The indications in addition to those mentioned above are absence of signs of severe myocardial degeneration and absence of obesity After operation the patient is allowed to become mildly myxœdematous at first, and then is given only the amount of thyroid extract that maintains a comfortable state of health This may be as little as five doses of gr $\frac{1}{2}$ per week In severe cases alcohol injection is also a measure to be considered but recently it has lost popularity If the theory of paradoxical action of diseased nerve endings is applied to thyroidectomy as in the theory of Raney s operation, the good results obtained can be easily understood for the thyroidectomy would in this manner reduce sympathectic tone and also reduce present or potential coronary spasm

Omentopexy has been an interesting experimental chapter in the surgical treatment of coronary disease It has not as yet advanced from that academic position I have had four cases thus treated, but am not convinced that revascularisation occurred in any of them

Differential Diagnosis

If the pain is quite typical of angina of effort in its position and its exactly proportional or quantitative relation to exercise, syphilitic aortitis, a recent small coronary infarct, and anæmia complicating slight loss of coronary elasticity must all be remembered and excluded Pneumothorax, especially on the left side may cause dyspnœa and pain on exertion, not unlike that of effort angina but radiation to the arm does not occur and the onset is probably very abrupt, moreover cough is not a coronary symptom X-ray screening or photography will reveal the cause of the symptoms Two fibrositic conditions may simulate angina of effort, intercostal fibrositis and left brachial neuritis The former pain is invariably definitely related to coughing, lifting, turning in bed, or other muscular activities The pain of brachial neuritis is usually fairly persistent, but is accentuated by exercise which involves moving the left arm It is associated with tingling and numbness, and even with detectable sensory changes, in the fingers, hand, or arm Dyspepsia is rarely mistaken for effort angina, though the effort angina, in cases where the pain is worse following meals, is often in the lay mind regarded as a form of dyspepsia

Finally there are cases with left thoracic pain, radiating to the left arm, and made worse after and not during exercise, which in its

acute form so closely simulates true anginal pain that cases are sometimes misdiagnosed as "angina" by physicians of repute To this pain various unsatisfactory labels have been attached in the past, such as pseudo-angina Left submammary pain is a good description of the milder cases, but does not seem adequate for the acute cases where the pain is severe and where radiation to the left arm is a prominent feature In view of the close simulation of effort angina and spasmodic angina, I have applied the somewhat controversial name "angina innocens" to this condition The chief diagnostic features distinguishing it from coronary pain are that its relation to exercise is not quantitative, for it follows rather than accompanies exercise, it is constantly accompanied by præcordial or apical tenderness and hyperæsthesia, sighing, sweating, tachycardia, dizziness and the other manifestations of neurocirculatory asthenia are often present It frequently accompanies mitral stenosis, but in the great majority of cases the heart is normal, [illegible] radiologically It occurs in [illegible] This condition is more fully [illegible]

Angina of Effort in Syphilitic Aortitis

In syphilitic aortitis the character of the pain and sense of constriction, its position, direction of radiation, and relation to exercise are identical with the same symptoms as they are seen in angina of effort due to coronary atheroma This is understandable for in both cases the lesion is a partial coronary obstruction The Wassermann reaction is positive in about 75 per cent of the syphilitic cases It is therefore necessary to focus attention on the clinical aspect so that syphilis may be suspected and treated in the remaining 25 per cent

In such cases angina of effort may appear without any other evidence of heart disease Such a patient may show absence of radiological enlargement, a normal or low diastolic and systolic blood pressure and a normal electrocardiogram Another group of cases presents evidence of enlargement and elongation of the aorta, either radiologically or by a loud aortic second sound, with a normal or rather low blood pressure and with no other evidence of arterial disease A third group provides a clearer hint by signs of aortic regurgitation especially if this is detected in a man of forty to fifty who is known to have been examined adequately for life assurance or for the Services some few years previously and passed as normal

In syphilitic aortic regurgitation the murmur is often heard better to the right of the sternum than to the left where the murmur is loudest in the rheumatic cases

This lesion must be treated adequately (see p 149) But the therapeutic risk is greater than if angina of effort were absent for some coronary ischæmia must be present Further occlusion may follow drug treatment either from a local intensification of the inflammatory œdema or in advanced cases from further fibrous shrinking of the aorta round the coronary mouths during the process of healing Nevertheless the disease can be arrested by adequate therapy in a reasonable percentage of these cases

CHAPTER XXX

LEFT SUBMAMMARY PAIN AND ANGINA INNOCENS

SINCE the days of Sir William Broadbent, who spoke of "pseudo-angina," an innocent type of pain has been noticed in individuals showing other cardiovascular symptoms and signs In many mild cases the pain is little more than a dull ache, but in others it is so acute that it is mistaken for a true coronary type of anginal pain The similarity is accentuated by its tendency to radiate into and down the left arm

Symptoms

The pain is left-sided and not central It is induced by exercise, arising during the stage of fatigue which follows exertion and is thus not quantitative to the amount of exercise taken It is nearly always accompanied by hyperæsthesia of the left chest, so that patients dislike sleeping upon their left side and male patients avoid carrying hard objects in their left breast pockets It often radiates down the left arm both in the mild and the acute types In the syncopal type this radiation often precedes the fainting attack Other signs of vaso-motor instability are present, such as variations caused in the pulse and blood pressure by the change from the recumbent to the standing posture, the pulse rate rising and the blood pressure falling on rising to the erect position Fainting and dizziness are common The patients are sensitive and nervous, and psychological examination reveals a percentage of mild psychosis above the normal figure

In addition to the ordinary persisting dull ache, sudden stabs of severe pain may occur These not infrequently are immediately followed by a syncopal attack This syncopal type of angina innocens is somewhat alarming to the patient, and also to the relatives, for both fainting and pain radiating from the chest to the arm are regarded by the laymen as signs of severe organic heart disease The sharp stabbing pain is often followed by a severe exacerbation of the dull left sided ache Whether the pain is so comparatively slight as to be adequately described as left submammary pain, or whether it is so severe as to suggest angina, and so to merit the term

angina innocens, it tends to be prolonged over a period as short as one hour or as long as a day or more

In the great majority of cases evidence of organic heart disease is absent, but, since organic disease attacks hypersensitive as well as phlegmatic individuals, cases of angina innocens are sometimes found in association with organic disease of the heart It is an easy matter in such cases to decide that the pain is not coronary, for it is not accurately proportional to exercise The types of heart disease in which this innocent or functional pain is not unusual are mitral stenosis, in which condition submammary pain is common and a few cases of *coronary infarction*, followed by *cardiac anxiety and excessive* heart consciousness Mitral stenosis induces heart consciousness mechanically, from the slapping knocking quality of the apical impulse against the chest wall

Signs

Physical examination of most cases of angina innocens or left submammary pain shows no cardiac abnormality The heart is normal in size, and the sounds are also normal The X ray picture and electrocardiogram confirm this opinion Unlike organic types of cardiac pain, this condition is associated with a hyperæsthesia of the chest wall, the tender area extending inwards from the *apex beat to the mid line, and* upwards from it over the left pectoralis major A spot of maximum tenderness is often found just at or within an inch of the apex beat, and can be often localised just at or near to the lowest part of the outer margin of the left pectoralis muscle where this is inserted into the fifth rib Exercise causes tachycardia out of proportion to the effort, as is shown by exercise tolerance tests The first sound is often unduly loud and slapping, but unlike that of mitral stenosis is low-pitched Functional murmurs are common Other signs of effort syndrome are often present

It has been suggested that angina innocens, or left submammary pain, has a fibrositic origin and character, but several points argue against this theory Fibrositis attacks the most used muscles, and most people are right-handed, whereas this pain never is The syncopal type of angina innocens is a *definite* entity, and no other fibrositis produces syncope Radiant heat and massage help fibrositis, but have no influence on this condition

Knowledge as to the true mechanism of production of the pain is lacking The important facts about it are as follows it is an

easily recognisable clinical entity, it is found typically in highly strung nervous people, the heart is organically normal, except in mitral stenosis, in which condition the pain is not uncommon, in severe cases it so closely stimulates true angina that mistakes are not infrequently made in diagnosis by competent physicians

Treatment

The treatment is exceedingly difficult and the immediate results very disappointing The first step is a clinical examination so careful and complete as to convince the patient that the verdict is to be trusted After this he or she can be assured that the heart is not diseased, that "angina" is not present, that the particular pain is a sign of an irritable or nervous heart, and that the condition is never found after the age of fifty or so Since patients do not die of the condition, *this latter statement must mean that they all recover from* it The next step is to explain that some time may pass before the symptoms disappear, months or even years, and that this chronicity is bound to be somewhat depressing, but the situation must be faced, and can be faced with confidence and optimism

The opinion of a psychiatrist should be obtained in severe or persistent cases, and considerable benefit to the patient may result from appropriate treatment A further essential therapeutic step is to make a thorough examination to exclude the presence of any physical abnormality which could in any way have deleterious effects on the general health, for example, definitely infected tonsils should be removed.

These investigations and measures having been undertaken the following rules of life must be followed Such exercise can be allowed, and encouraged, as does not induce excessive fatigue or accentuate the pain As much sleep, or rest in bed at night as possible must be enjoyed Many such patients are able to carry on with their work, provided they get to bed early enough every evening All removable causes of worry and of mental fatigue must be eliminated Apart from sedatives, which help in some cases, drugs are ineffective

Diffuse infiltration of the left pectoral region by subcutaneous and intramuscular injection of one per cent novocaine is worth trying, but this measure often fails Deep X-ray treatment to the left first five dorsal sympathetic nerve roots does not help Deep massage and short-wave therapy to the area of pain fails to give relief These therapeutic failures are mentioned because in so persistent

a condition the knowledge of what treatment to avoid is nearly as useful as more positive information The most helpful single therapeutic step is to convince the patient that the pain is not due to any form of organic heart disease, and that it will certainly in time disappear

CHAPTER XXXI

COR PULMONALE

In hypertension the raised systemic arterial blood pressure places an increased burden upon the left ventricle If the left ventricular muscle becomes diseased or deprived of oxygen it fails, suddenly or gradually, according to the rapidity of development of the disease or the anoxæmia In a far smaller number of cases similar vascular and cardiac changes may be entirely restricted to the pulmonary arterial circulation and the right ventricle When the right ventricle alone fails in this manner, the clinical condition is labelled acute or chronic ' cor pulmonale,' according to the speed of onset

Acute strain upon, and failure of, the right ventricle alone occurs most commonly in extensive pulmonary embolism, secondary to phlebitis Here not only is the pressure in the pulmonary arterial system abruptly increased, but shock and anoxæmia are also added In severe pneumonia a somewhat similar picture is produced

Since the right ventricle is much weaker than the left it feels the effects of a suddenly imposed strain more quickly and more severely A chronic strain is at first supported reasonably well by the right ventricle, but for a shorter time than by the stronger left chamber Chronic right sided failure is most often the result of either chronic fibrosis of the lungs, or of changes in the pulmonary arterioles analogous to those in the systemic arterial system in hypertension

ACUTE COR PULMONALE

Phlebitis in the veins of the legs or of the pelvis, is the usual source of the massive clot responsible for acute pulmonary hypertension The thrombus abruptly becomes freed locally, and is flushed through the vena cava and right heart into the pulmonary arteries If the clot is small, and invades only a small pulmonary vessel, the impediment to the function of the right ventricle is insufficient to cause severe symptoms or signs But if the clot is large the condition is serious and acute

Since younger people do not get phlebitis acute cor pulmonale' is rarely seen below the age of forty Any condition causing stasis in the peripheral circulation, surgical gynæcological, or medical may be responsible for clot formation in the calf veins An actual phlebitis need not be present Not until recently has it become known that thrombosis due to stasis is common in general medical conditions and is a serious complication in many cases of chronic cardiac disease

There may be no clinical evidence of phlebitis on clinical examination of the legs Unexplained febrile attacks, tachycardia, pulmonary *signs suggesting transient and mild broncho pneumonia*, cough, pleurisy, or slight hæmoptysis may hint at the deposition in the lungs of small emboli The X-ray may show either recent local infarction, or the linear scars of old infarcts But the sudden advent of a big embolus is the cause of acute cor pulmonale

The **symptoms** are abrupt in their onset Great respiratory distress is present, with increase in the speed and depth of respiration Cough may be definite A sense of oppression is usual and this in some cases is increased to pain across the chest Shock is rapid and profound and the heart rate is often much increased more so indeed than may be found in a case of coronary infarction with a corresponding degree of pain

The **signs** most commonly apparent are as follows dyspnœa, sweating, pallor and tachycardia, clinical evidence of the distended pulmonary artery, such as palpable pulsation under the firmly placed hand in the second and third left spaces near the sternum, an increase upwards of the area of cardiac dullness, an accentuation *of the pulmonary second sound or a systolic pulmonary murmur*, there may be evidence of sudden systemic venous congestion, shown by distension of the veins in the neck, a sign which may be cancelled out if the associated shock is severe enough, and lastly changes in the electrocardiogram may occur Both the acuteness of the symptoms and the electrocardiographic *abnormalities are transient* In Lead 1 the S wave is present, or deepened, in Lead 2 the T wave is flattened or inverted, in Lead 3 a deep Q wave is found and there is S-T elevation, in Lead 4 the T wave is depressed

The **treatment** of the acute phase is rest, oxygen, and morphia But the prophylactic treatment is of primary importance Every medical case, including patients with chronic heart failure or coronary infarction, should be made to exercise the feet and lower legs twice daily, throughout the period of rest in bed If this were done no only would a majority of cases of acute cor pulmonale never oc

but in addition many rather indefinite pulmonary complications, in reality due to minor pulmonary embolism, would be avoided, and the number of deaths from unexplained complications during prolonged illness would be reduced

CHRONIC COR PULMONALE

Chronic cor pulmonale is uncommon Its two most typical manifestations occur as a result either of pulmonary arteriosclerosis or chronic pulmonary fibrosis

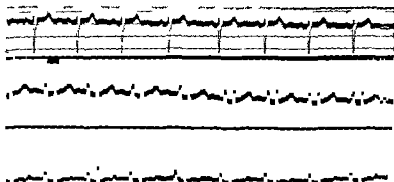


Fig 63—Typical Right Axis Deviation

The S wave is deepest in Lead 1 and the R wave is tallest in Lead 3 There s some evidence also of auricular hypertrophy in the tall peaked P wa es in Leads 2 and 3 The pat ent had an advanced degree of emphysema and chron c bronchit s and was beginning to show evidence of early myocardial fa lure w th signs of system c congest on

Pulmonary arteriosclerosis (Ayerza's disease) is a rare but striking condition *in which progressive arteriosclerotic changes in* the peripheral pulmonary arterioles interfere with the proper oxy genation of the blood and throw an ever increasing burden upon the right ventricle The cause is unknown The age incidence lies between thirty and forty but younger cases are met with The patient complains of increasing shortness of breath and in the end may develop œdema hepatic and renal congestion and other signs of failure Simultaneously there is a progressive cyanosis due partly to slowing of the circulation rate and partly to a compensatory erythræmia for the red blood cell count often increases to six or seven millions or more with corresponding figures for the hæmo globin The right ventricle becomes progressively hypertrophied and its thrust may be felt in the epigastric notch Clubbing of the

fingers is often present A most notable peculiarity is the absence of orthopnœa, and this contrasts strongly with the dyspnœa orthopnœa, cyanosis and obvious distress of left heart failure There is no pulmonary congestion Right axis deviation in the electrocardiogram is invariable The treatment is palliative only The outlook is bad

Chronic pulmonary fibrosis due to silicosis or due to chronic bronchitis and emphysema, may also cause a progressively increasing strain upon the right side of the heart Chronic cor pulmonale from bronchitis is usually seen in elderly patients and the symptoms, predominantly those of the causative disease are dyspnœa and winter cough with some spasm of bronchioles But in addition there is increasing cardiac dyspnœa, as the myocardium loses its efficiency

Physical examination may reveal slight cyanosis a poorly expanding chest, with a wide subcostal angle, some clubbing of the fingers, and a palpable right ventricle Expiratory rhonchi are common [illegible] The red blood count may [illegible]

Treatment is of the causative condition An autogenous vaccine from the sputum is worth a trial Ephedrine may help An elastic abdominal support may increase the diaphragmatic excursion Digitalis benefits some patients Intercurrent pulmonary infection must be vigorously treated by oxygen tent, penicillin, or sulphonamides

CHAPTER XXXII

ANEURYSMS

THE aorta is the usual site of the local arterial dilations caused by syphilis or atheroma, and also of the undermining and splitting process which is called dissecting aneurysm

The arteries of the periphery are more commonly weakened by infected emboli so as to stretch locally into the form of mycotic aneurysms They are also more likely to be injured by external wounds involving the artery and its adjacent vein Such an injury thus produces a local swelling in which these vessels anastomose, which is called an arteriovenous aneurysm

ANEURYSM OF THE AORTA

Syphilis and atheroma are the two causes of aortic aneurysm The incidence of syphilitic disease is greatest at the cardiac end of the aorta, and that of atheroma at the iliac end of the vessel Most intrathoracic aneurysms are therefore syphilitic, and most abdominal aneurysms are atheromatous Syphilis causes the great majority of all aortic aneurysms Destruction of the elastic tissue of the aorta is *the chief cause of syphilitic aneurysm, for the* fibrous tissue which replaces it retracts poorly and ultimately stretches

Syphilitic disease of the aorta is an actively progressive process, and the presence of an aneurysm, therefore, indicates both a severe and an advancing lesion Atheroma however severe, is more passive and is not inflammatory, so that an atheromatous aneurysm in the abdominal aorta may remain unchanged for years after its formation

An aneurysm may bulge locally, or may constitute a gradual and uniform stretching of the vessel The former is called a saccular and the latter a fusiform aneurysm Syphilitic aneurysms are often multiple, and since they are chiefly found in men between fifty and sixty years of age the local lesion is frequently complicated by much atheromatous change in addition

The dangers to a patient of an aneurysm are various The mass

of the swelling may press upon bronchi œsophagus, and veins causing respiratory embarrassment, dysphagia or circulatory dis order It may stretch the aortic ring causing aortic regurgitation, or may involve the coronary orifices, producing coronary pain or heart failure Or the wall may rupture into the pericardium lung, or œsophagus, or through the skin

An aneurysm of the aorta does not by itself cause enlargement or other change in the heart, a backwater has no obstructive effect on the main stream The aortic second sound is often accentuated because the aortic surface is nearer to the stethoscope then normal Aortic regurgitation caused by an aneurysm has the usual characteristics

The symptoms and signs vary greatly with the position of the swelling From the *ascending aorta* the aneurysm usually spreads forwards towards the anterior surface of the right chest It causes little embarrassment to vital activities, but when it meets the ribs and sternum it presses on nerves and on bone and causes pain It soon becomes obvious through the production of a uniform pulsating swelling which is seen and felt chiefly in the third, second and sometimes the fourth right interspaces Pulsation is most visible in a good oblique light, and best felt by firmly compressing the right chest between both hands as the patient sits or stands The pulsation can be shown to be expansile by placing two fingers firmly one on each opposite slope of the tumour The percussion note is much impaired over the aneurysm Breath sounds are diminished or absent A thrill and a murmur, usually systolic, may or may not be present The aortic second sound is often accentuated, and may even be "palpable", it is then called diastolic shock The aneurysmal swelling may become a large prominent pulsating mass in the right chest surface

Aneurysms of the first or ascending part of the aorta have further potentialities A small unsuspected swelling may point inside the pericardium, its rupture causing sudden unexpected death Another aneurysm may point towards, and press upon, or even rupture into, the superior vena cava, causing increasing distension and engorgement of the veins and tissues of the head face, neck and arms, and upper chest These signs are first gradual, when they are caused by pressure only, but are suddenly accentuated if rupture occurs into the vena cava Other aneurysms of this part of the vessel often involve the aortic ring, causing aortic regurgitation One interesting case was that of a man whose aneurysm pointed rather low, over the third and fourth right spaces, and yet had not produced aortic

regurgitation These facts indicated that it was very saccular, a point which was confirmed radiologically The aneurysm was therefore wired, a Colt's wire being inserted into it under local anæsthesia The expansile pulsation ceased and the swelling became much smaller for a year It ultimately increased again in size and the patient died

Aneurysms of the arch of the aorta, being deeply placed in the thorax, are close to a number of sensitive and vital structures They have therefore been called *aneurysms of symptoms* Pressure on the left recurrent laryngeal nerve, as it loops round the stretched aorta, causes hoarseness and a clanging, noisy cough from paralysis of the left vocal cord Pressure on the trachea or on a main bronchus causes dyspnœa and a muffled cough, and later may produce bronchiectasis or may leak or rupture into the air passages One such small aneurysm pressing on the left main bronchus gave rise to severe orthopnœa and dyspnœa reminiscent of an attack of *acute asthma It was invisible radiologically, but an obstructive* cough and a positive Wassermann reaction gave the clues to its presence

The pulse wave down either brachial artery may be delayed in time or diminished in volume, if the vessel's mouth is involved in the aortitis of the aneurysmal wall, or in the clot of the aneurysmal sac The blood pressure should be taken in both arms when an aortic aneurysm is suspected

Pressure on the sympathetic nerves, *usually the left*, may at first stimulate but will soon paralyse these structures Preliminary retraction of the lid, with an enlarged pupil, is soon followed by Horner s syndrome, enophthalmos a dropped eyelid, and a diminished pupil One such case was that of a naval rating, who was engaged in coaling stripped to the waist He was noticed to be black everywhere except over his left upper chest and back and left arm His sweating was locally diminished over this area by sympathetic paralysis caused by an aneurysm

Some aneurysms of the arch point backwards and to the left, causing collapse of the left lung, or even bronchiectasis, and eventually may become manifest in the left interscapular region as large pulsating swellings Others involving the innominate artery travel upwards and to the right, collapsing the right upper lobe and simulating carcinoma of the right upper bronchus A useful diagnostic point is that in such a case the trachea is deviated away from the bulky aneurysm, but is drawn towards the lung-shrinking carcinoma

Aneurysms of the descending aorta are rarer than the above two types, but have certain distinctive features When it lies at the level of the ventricular part of the heart the vascular swelling is situated behind that of the ventricles The œsophagus is between these structures Since heart and aneurysm stiffen simultaneously with the force of systole, pressure is exerted by both laterally and forwards against the left chest and backwards laterally against the vertebral column The consequences are that the vertebral bodies quickly become eroded, causing pain in the back the œsophagus is nipped, producing dysphagia, and the chest as a whole is pushed to the left with systole causing "lateral thoracic jerk' This systolic jerking of the chest can also be produced by a large left ventricle, or by the presence of greatly enlarged auricles behind strongly beating ventricles, as in some cases of mitral disease When an obvious lateral thoracic jerk is found in an adult of about fifty, and with a ventricle clearly not enlarged the presumption of aneurysm of the descending aorta is strong The sign in such a case has stimulated me to look for, and enabled me to find on the X ray film, an aneurysm which the radiologist had missed, behind the cardiac shadow

Diagnosis depends on careful consideration of the symptoms and signs already described Difficulty often occurs in distinguishing aortic aneurysm from bronchial carcinoma The displacement of the mediastinum is a helpful point An aneurysm is apt to push the trachea away to the opposite side, whereas a bronchial carcinoma is more prone to collapse the lobe of the lung and draw the trachea to the same side Pain as a rule, is earlier and more severe in aneurysm than in bronchial carcinoma In some cases these points are more helpful than is the X-ray picture But radiography provides in most cases, the confirmatory evidence as to the presence of an aneurysm (Figs 64, 65) The aneurysmal shadow may be missed either by being small and centrally placed, or by being behind the heart's shadow The Wassermann reaction is positive in about 70 per cent of cases of aneurysm of the aorta Thus a negative result does not exclude the condition

The **treatment** is identical with that described under syphilitic aortic regurgitation An occasional saccular aneurysm is worth treating surgically by the insertion of wire through a cannula A presenting aneurysm, of the ascending aorta, but with no associated aortic regurgitation, would be a case in which wiring should be considered It is worth while instituting and completing a thorough

course of antisyphilitic therapy but the results are less good than in cases of aortitis without aneurysm

Any deterioration in the clinical condition during antisyphilitic

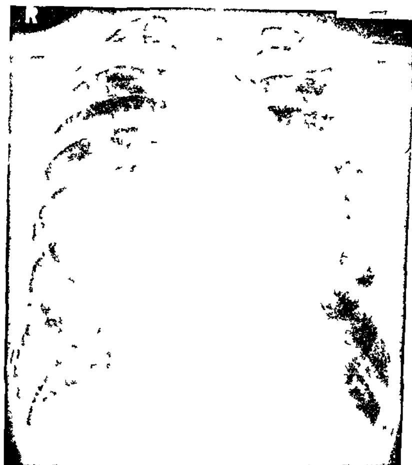


FIG 64 —ADVANCED SYPHILITIC AORTITIS WITH ANEURYSM FORMATION

An aneurysm is present extending from the ascending aorta to the right The aortic knuckle is much enlarged A second aneurysm is extending to the left In the right lower and mid lobes a honeycomb shadow is seen typical of bronchiectasis The left ventricle is not enlarged

treatment must be most carefully watched because aneurysm is a rather late phase of the disease and there is a likelihood that fibrosis to a considerable extent may be present round the coronary orifices If therapy is clearly benefiting the patient and if no coronary pain or

increased dyspnœa supervenes the coronary orifices can be considered reasonably free from disease But if such symptoms become

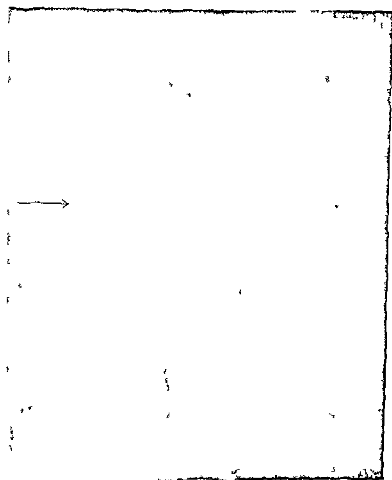


FIG 65—RIGHT ANTERIOR OBLIQUE SAME PATIENT AS FIG 64

prominent during drug treatment the therapy may be speeding up fibrosis round the coronary mouths and irremediable harm may be done

Should no increasing dyspnœa or anginal pain accompany

antisyphilitic treatment there is some chance that this may arrest or at least slow down, the disease Most cases of syphilitic aneurysm do not live five years but in a few the disease becomes stationary and this span is considerably exceeded

ABDOMINAL ANEURYSM

The majority of abdominal aneurysms are due to atheroma They appear as pulsating, roughly spherical swellings just to the left of the mid line in the upper or middle abdomen The pulsation can generally be felt to be expansile The only symptom is slight discomfort occasionally increased to pain There are no pressure effects Atheroma usually of an advanced degree, is the cause and the X-ray is apt to reveal calcification both in the aorta and in the wall of the aneurysm

The condition most often to be distinguished from it is a pancreatic cyst In this condition the pulsation is transmitted and not expansile, and the position is often more to the left of the mid line The younger the patient the more likely is the swelling to be a cyst There may be a history of gall stone colic

There is no active treatment for most abdominal aneurysms though in some cases wiring is a possibility I have known one remain unchanged for ten years

DISSECTING ANEURYSMS

Dissecting aneurysm of the aorta is an uncommon condition but is less rare in proportion to the diligence with which it is looked for, especially post mortem

It seems to start as a form of local degeneration in the media and over this the intima ruptures allowing the aortic blood to force its way into the medial or adventitious aortic layer Atheroma is the commonest associated lesion syphilis as a cause is rare Many patients have previously had a raised blood pressure from hypertension, and a few coarctation of the aorta

The blood usually splits its way in both directions, up towards the heart and down towards the diaphragm The upper infiltration is usually shorter than the lower, and may be absent The lateral extent of the split varies, but may be up to three quarters of the circumference of the vessel The blood tracks onwards at a greatly varying speed and may find its way back either into the pericardium, or laterally into the mediastinum, the pleura, or the pulmonary

tissue, or onwards down the aorta The pericardial pleural and pulmonary leaks are fatal, the former in seconds and the latter two in hours or days

When the lesion has extended as far as the diaphragm the blood may find its way back to the normal channel and in rare cases the false aorta may become stabilised and continue to function for months, or even years The return to the normal channel is occasionally even further down beyond the diaphragm

The **symptoms** in many cases are characteristic The onset is abrupt, the patient, who nearly always has chronic hypertension, feeling that he was hit in the back From the first the pain is extremely acute, not building up slowly to a maximum as in coronary infarction The pain is most often behind, but may radiate to the front, to the neck, or down to the thighs or loins Fever may be present Collapse of a profound degree often occurs with pallor, sweating and a great fall in blood pressure Death may follow quickly if the leak extends to pericardium or lung

In other cases two or more severe attacks occur, without the collapse, as preliminary incidents These may precede the final severe attacks by weeks and are presumably due to earlier and lesser grades of dissection Shortness of breath is not a prominent symptom

On examination, the **signs** are often inconclusive The blood-pressure fall is often great, but the tension may rise again as the patient recovers from the initial shock The pressure in the legs may be greatly reduced if the swelling caused by the dissection encroaches on the aortic lumen Should the proximal ends of limb arteries be involved in the lesion, the blood pressure in one or both arms, or in a leg, will be found to be lowered The other signs are very variable Pleural or even pericardial friction may be detected Râles may be present over one lung only resulting from mediastinal interference with the blood flow to or from that organ The venous return from neck or arm may be visibly impeded

X-ray examination may reveal no discoverable aortic abnormality, but the vessel may be widened or unusually high in the thorax, or a shadow may extend from it in the direction of the innominate or the left subclavian artery The electrocardiogram is typically normal, but should the aneurysm travel back to the level of the coronary arteries changes suggesting coronary infarct may be produced The sedimentation rate of the blood may be raised

Treatment consists in controlling the pain by morphia, and by resting the patient in bed, in the hope that recanalisation back into

the lower aorta is occurring Should this be achieved there is some hope of survival for a period

ARTERIOVENOUS ANEURYSM

This condition is the result of an anastomosis between an artery and the adjacent vein Trauma is the commonest cause, and the vessels of the periphery provide the most usual site for the lesion The effects of the leak are similar to those of aortic incompetence, or of a wide patent ductus arteriosus The diastolic pressure is much lowered, and hypertrophy and dilatation of the left ventricle occur The treatment is surgical repair of the leak, after which the cardiovascular abnormalities disappear Paget's disease may cause, in the bone, a species of arteriovenous aneurysm

Section VIII

CHAPTER XXXIII

THE HEART IN PREGNANCY

THE pregnant woman with heart disease faces three distinct physical hazards She must surmount the biological and the mechanical difficulties imposed on her body by the presence of the growing fœtus within the abdomen She must put out the muscular effort needed for the expulsion of the baby on coming to term She will have the subsequent mental and physical burden attendant upon the nursing and the bringing up of an additional child These three problems must each be considered separately in the individual case

A normal pregnancy affects the work of the heart in three ways the growing fœtus needs oxygen and food, and demands the removal of waste products, the maternal thoracic capacity and diaphragmatic excursion are both diminished as the uterus swells, so that inspiration is less efficient and the heart is pressed up from below, the free arteriovenous anastomosis in the placenta introduces to some degree into the maternal circulation the mechanical effect of an arteriovenous aneurysm

Additional handicaps to an efficient cardiac response to pregnancy may be added, either by non-cardiac pathological conditions, or by organic cardiac disease Anæmia is probably the commonest non-cardiac example, and its proper treatment is of the greatest importance, whether organic cardiac disease is present or not The effects of an organic heart lesion are greatly increased by severe anæmia The three commonest forms of heart disease in pregnant women are rheumatic carditis, congenital cardiac lesions, and thyrotoxicosis The age incidence of myocardial degeneration, and of hypertension, is on the whole so late in life that these rarely complicate pregnancy

Pregnancy with Rheumatic Carditis

Mitral stenosis is the important lesion in the great majority of cases of heart disease complicating pregnancy Aortic regurgitation, also rheumatic, may coexist, or may occasionally occur alone The aortic lesion is often so small as to cause no clinical hypertrophy of

the left ventricle, and no lowering of the diastolic blood pressure In such cases the aortic diastolic murmur may be ignored, attention being entirely fixed upon the effects of the mitral disease Cases of mitral stenosis may be classifiable under various headings The first group comprises those with no symptoms, with no increase in cardiac size, and with no evidence of active acute rheumatism Such cases can be safely allowed to continue with their pregnancy and in all probability will achieve a satisfactory puerperium They must be closely watched If rheumatic fever supervenes, or definite evidence of active acute carditis, termination must be carefully considered

The second group consists of those women with no symptoms of heart failure, but with some slight increase in heart size These cases also may be allowed to continue If symptoms of slight failure appear, the patient should be put to bed and watched Here again a satisfactory pregnancy may be completed

A third group includes patients with more enlargement of the heart as a whole, the greater the enlargement the less safe is the pregnancy Thus great enlargement, even without serious symptoms, usually constitutes an indication for termination Definite symptoms of early failure, even without congestion, constitute another serious warning, and if present should suggest termination It is clear that the exact evaluation of the relative importance of symptoms of failure and of increase in heart size in any given patient is an affair of expert assessment and decision Rare cases with a theoretically poor outlook unexpectedly weather the storms of pregnancy But great responsibility attaches to the doctor who, with his eyes open, plans such a course

If a patient has had dyspnœa on exertion before pregnancy, or has had a physical setback during a former pregnancy, or has had definite congestive failure, or has auricular fibrillation, pregnancy is likely to cause a serious if not a fatal cardiac breakdown Pregnancy is also dangerous if aortic regurgitation, sufficient to have produced a low diastolic pressure, and palpable left ventricular hypertrophy coexist In all cases where there is a reasonable chance of a viable child, but where also there is a measurable risk of temporary or permanent cardiac deterioration, no decision should be reached without a full and frank discussion and explanation with both wife and husband

The next point to consider is the timing of the clinical decision This is determined by the fact that patients stand uterine evacuation badly after the end of the third month, so that the position must be

assessed and the programme laid down before that time Should a patient pass this period safely but begin to get symptoms or signs of failure subsequently, the wise course is to put the patient to bed, and to treat the heart failure as such leaving the pregnancy to continue to the time at which a viable child is to be expected

The desirable method of delivery of the cardiac case varies with different physicians I prefer Cæsarian section, for all cardiac patients stand anæsthetics well, provided that these are well given, and that the slightest anoxæmia is avoided Sterilisation may be done at the same time

The following case illustrates some of the problems arising in a case of mitral disease with pregnancy A primipara of twenty-four, pregnant two months, was seen in the outpatients' department to assess the safety of pregnancy She was very desirous of a child She had no symptoms of failure, and no tachycardia The heart was definitely somewhat enlarged The rhythm was regular the chief murmur present was that of mitral regurgitation A distant diastolic mitral murmur was audible in the left lateral position She continued well until the end of the fifth month, when dyspnœa *increased rather rapidly, orthopnœa appeared, and œdema of the* legs and feet occurred Three days after admission to hospital hæmoptysis commenced, and lasted eight days This was considerable, and the hæmoglobin fell to 52 per cent She simultaneously developed a right sided pleural effusion, and 200 c c of serous fluid were aspirated She remained in a very precarious state for several weeks and it was decided to treat the general condition, allowing the pregnancy to continue A week after the paracentesis seven transfusions of packed cells were given, extending over a period of thirteen days The total quantity of concentrated blood infused was 1,050 c c , the equivalent of 2,100 c c of whole blood Ferrous sulphate by mouth was also administered These measures raised the hæmoglobin to 90 per cent during the subsequent four months Digitalis did not appreciably help, for the rhythm remained regular throughout Oxygen was given during those periods when pulmonary œdema was pronounced A great factor in the patient's recovery was her continued courage, and the determination to have her baby, of which she was delivered by Cæsarian section shortly before term She was ambulatory, and without congestive symptoms, one year *later, and dyspnœa on ordinary walking was slight*

The effect of pregnancy upon other cardiac lesions can be summarised briefly as follows, but this summary can only be regarded as a succession of generalisations, since every case

separate and full individual consideration Aortic regurgitation alone is no bar to a successful pregnancy if the leak is slight, a considerable or a great reflux constitute dangers in proportion to the extent of the lesion In aortic stenosis the risk generally is not great In pure mitral regurgitation the risk is slight The acyanotic varieties of congenital heart disease stand pregnancy well But I have known a case of symptomless congenital heart block die suddenly and unexpectedly in childbirth Previous coronary thrombosis constitutes a risk requiring expert evaluation, it is not necessarily a contraindication

As regards the irregularities occurring alone, and without evidence of myocardial disease, both paroxysmal tachycardia of the youthful type and auricular and ventricular premature beats can be ignored Long and severe attacks of paroxysmal tachycardia may be dangerous to the expectant mother for they not only may be exacerbated by pregnancy but the enforced use of quinidine is very apt to cause abortion

CHAPTER XXXIV

EFFORT SYNDROME

EFFORT syndrome, or neurocirculatory asthenia, is the label applied to a condition of hypersensitivity both of the heart and of the nervous system The title 'Effort Syndrome' is an apt one, in so far as the symptoms complained of by these patients after very little exertion are much the same as those noticed by normal individuals after considerable exertion The title neurocirculatory asthenia is also applicable, for the condition is both nervous and circulatory and the outstanding symptom is fatigue or asthenia

Symptoms

The symptoms are exhaustion dyspnœa, palpitation, sweating, heart-consciousness, præcordial discomfort or pain, dizziness or faintness, tremulousness and nervous irritability The patients are generally young adults and the condition is unusual after the age of forty-five and rare after fifty five It is practically unknown in children

The determining factor in the production of the syndrome seems to be the character of the individual nervous system Highly reactive, sensitive persons are more prone than others to the disorder Occasionally there is a psychoneurotic basis

Infection sometimes plays a part, removal, for example, of grossly infected tonsils or of amœbic infection, may produce a rapid disappearance of the symptoms Prolonged worry and strain are the chief causative factors in other cases

The advent of organic heart disease, or the fear of organic heart disease, whether this is present or not, is in others the starting-point of "effort syndrome," the symptoms of which may persist after recovery from the organic disease

Effort syndrome is often a very chronic condition, and although the duration may be one of months only, it may be prolonged to five or ten years The most prominent symptom is great fatigue, mental and physical, and this is usually associated with the cardiac phenomena palpitation, dyspnœa, and left chest pain A valuable

point to elucidate is the relative severity of lassitude and dyspnœa In "effort syndrome" the exhaustion is more complained of than is the shortness of breath, whereas in organic heart disease the reverse is the case It seems that the nerve supply to and from the heart is much more sensitive than normal so that the motor effects of exertion and emotion produce an undue acceleration of the heart, and the sensory impulses so produced render the individual unduly conscious of the heart's action whether in exercise or at rest

Palpitation is always present Heartache may be slight but is often severe enough to merit the label "angina innocens" A full clinical description of this aspect of "effort syndrome' is to be found in Chapter XXX

The dyspnœa is of two varieties, and is either of the normal variety occurring after exertion, or is in reality a nervous 'lung *consciousness" This latter makes the patient feel that the chest* will not hold enough air and causes him or her to sigh deeply and frequently 'I can t seem to get enough air into my chest" is the usual complaint

Giddiness and faintness or fainting may also occur, and are vasomotor in origin being often related to posture and increased by standing Standing, particularly for long periods, is especially trying and exhausting for these patients Finally nervousness or jumpiness is often complained of

Signs

The heart is typically normal, and is often small in relation to the patient's height weight and age, and is frequently of the narrow, long, vertical type

The commonest form of organic heart disease in which "effort syndrome is also found is mitral stenosis

The resting heart rate is usually normal, when it can be recorded without exciting the patient's nervous system, but physical examination nearly always causes it to be increased, and after moderate exercise this increase becomes excessive

Exercise tolerance tests, which consist of counting the heart rate *before, during, and after a measured* amount of exertion, are thus more sensitive indicators of this condition than they are of diminished efficiency in a diseased heart The presence of effort syndrome, even of a slight degree, will thus interfere with any conclusions which might otherwise be drawn from such tests, the cardiac value of which is problematical

The cardiac action is often vigorous, and by contrast the pulse

volume seems to be small This cardiac overaction sometimes suggests falsely the presence of a thrill

Sweating is common, but this is a cold sweat, and is a localised phenomenon, usually confined to the axillæ, palms forehead, or neck in contradistinction to the generalised warm moist skin of thyrotoxicosis A coarse tremor may be present That of thyrotoxicosis is usually fine

The reflexes are exaggerated Vasomotor instability is often considerable This is best shown by a variation in the heart rate and blood-pressure figures as between the recumbent and the standing positions

Normally the heart rate is little if at all increased by standing, *and the blood pressure little if at all changed But in effort syndrome* recumbent heart rate may rise from 80 to 120 when the individual stands up, and a recumbent blood pressure fall from 140/80 to 110/72 Both the heart rate and the blood pressure should be measured not immediately the patient stands up but after he has been upright for two minutes

Diagnosis

Lassitude, palpitation, and left chest ache in a young sensitive person are pathognomonic of effort syndrome The differential diagnosis most commonly lies between it and such complaints as paroxysmal tachycardia, early thyrotoxicosis, and anæmia The latter is excluded by blood examination, the former two problems are dealt with in Chapters XIII and X

Coronary disease may be suggested when the left chest pain is acute, for in both conditions the pain may radiate down the arm This question is fully dealt with in Chapter XXX

Treatment

There are three essentials in the treatment of effort syndrome a very complete investigation of the case, a sympathetic attitude on the part of the physician, and a firm opinion that organic heart disease is not the cause of the symptoms and that recovery in time is certain

In addition, all definite foci of infection or sources of physical ill health must be removed Rest is essential but this, it must be explained, is for the nervous or general condition and not for the heart Patients when sitting should have the legs and feet raised

Reassurance is justifiable, for cases after fifty years of age are rare, and the patients do not die, recovery therefore will inevitably occur On the other hand, patience will be necessary, for the duration of effort syndrome is often long If the patient's impatience should induce over-exertion much above the capacity of the moment a relapse is liable to occur

Increases in exertion and exercise should therefore be gradual, but definite, and must remain always below a certain level, found by experience, beyond which overfatigue and an exacerbation of symptoms are caused, and below which general improvement is slowly gained

Help may in some cases be obtained from a psychiatrist General massage is sometimes helpful Sedatives are often useful Valerian is valuable in a few cases

CHAPTER XXX

CARDIAC SIGNS IN SYMPTOM FREE NORMAL *YOUNG ADULTS*

DURING the six years of war (1939 45) a large number of young adults of both sexes were examined cardiologically by the author at the request of various medical boards About a quarter of these young people, 308 in all, were analysed carefully and this analysis is the basis of the present chapter 224 had normal hearts and in 84 organic heart disease was present without symptoms

It is clear that the cases were selected by the boards, which were three in number, and which were composed of a number of different medical practitioners The extent and type of this selection clearly varied from board to board, and from doctor to doctor, so that the results described are not a satisfactory study of the distribution of such signs in the normal population But the study probably gives a true picture of the sort of patient who is usually suspected by a doctor of having some cardiac abnormality

The method of procedure was first a full clinical examination, and then radiological investigation in the antero posterior and the right and left oblique positions Where the pulmonary conus was unusually prominent a barium swallow was also done, and the size of the left auricle thus carefully checked The electrocardiogram was not taken as a routine, since it was found quite early in the investigation of these recruits that it yielded little additional information, except in selected cases

When young people are examined from the point of view of the
… be reached Either the

finally, both function and physical state are both deficient The first and the last of these possibilities do not raise any question as to the state of the heart The second group contains individuals with what is usually known as "effort syndrome" There remains the third large class in which signs suggesting some cardiac abnormality are present, in the absence of any symptoms This is the

group in which diagnosis is frequently difficult, but which is from the point of view of the State very important, because admission to the Services or to any pensionable position is likely to involve expense if an individual with organic heart disease is accepted These slight abnormalities, or suspected abnormalities, are described below They fall into certain well defined clinical groups

The size of the heart is the most important single fact in the investigation of a cardiac patient If the heart is enlarged it follows as a matter of course that it must be regarded as diseased, whether such disease be active or not If the heart is normal in size it may be concluded especially in a young individual that disease is probably absent If the heart is of the small longitudinal type, well below the average, no disease of an organic nature is present

In many healthy but excited young adults the position of the apex beat is deceptive This especially in athletic individuals, is frequently so forcible that it seems to be in the mid-clavicular line, or external to it Here an accurate radiological examination is the only means of determining whether the heart is enlarged or not There are two methods a six foot film, or measurement by orthodiagram Of the two, the latter is probably more accurate, since it is possible to measure the heart either in systole or in diastole as is desired The vigorously beating heart above described is found by such measurement not to be enlarged, in spite of the apparently abnormal position of the apex beat

The heart sounds which are described in the following paragraphs were present in every case in young adults, whose hearts were regarded as being normal both clinically and radiologically Moreover in all these cases the blood pressure was within normal limits Such functional or physiological murmurs and unusual sounds may also, of course, be present together with the murmurs of organic valvular disease These heart sounds introduce many difficulties into the problem of diagnosis Abnormalities occur in the nature of the first and second normal heart sounds, and also in the appearance sometimes of a rather loud physiological third heart sound Furthermore murmurs and adventitious sounds are very common and these in their turn present problems

The first heart sound is frequently muffled or impure This is particularly the case when it is loud and when the heart is beating excitably or vigorously It may occasionally be reduplicated The most important single point about the first heart sound is its musical pitch It may be stated that when the first heart sound is loud but low pitched it is normal, and when the pitch is raised, approximating

more to the quality of the second sound, there is suspicion of abnormality

The second sound *is usually investigated at the base of the heart* Reduplication is present fairly frequently at the pulmonary base, and is of no especial significance Such reduplication may vary with respiration, chiefly appearing during inspiration

The third sound is a physiological phenomenon, but it is not very easily heard and is present only in a small percentage of cases It was heard in about 15 per cent of the cases here described It is audible at the apex and is a single distant sound It is heard after the second sound, which it follows at the same interval in any particular case It is frequently more audible in the left lateral position and in the last half of expiration During inspiration it often disappears It is more easily heard when the heart is beating slowly This sound is important, for it may be loosely described as a mid-diastolic murmur and may wrongly suggest mitral stenosis The points to be stressed are that the sound is short and detached, and is associated with no presystolic murmur and with no distant diastolic rumble Moreover the first sound is of normal pitch, whereas in mitral stenosis the pitch is raised X-ray examination of such patients frequently reveals a small heart less even than the normal size for the patient's age and physique, and with no increase in the size of pulmonary conus A third heart sound may also be heard in diseased hearts, particularly where there is ventricular dilatation secondary to hypertension, also in some cases of mitral stenosis, and finally in patients with an early stage of heart block In the hypertensive cases the additional sound is related in time to the first sound and not to the second In mitral stenosis the diastolic murmur may suggest a third heart sound but there is usually a definite underlying distant diastolic rumble also present, especially in the left lateral position and after exercise After the first stage of heart block *where the P–R interval is prolonged, auricular systole may become* unusually audible It is clear that the cases of physiological third heart sound described above belong to none of these three groups

There are a variety of murmurs and abnormal signs which may give rise to difficulty in diagnosis First there are the *systolic murmurs* The systolic murmur heard *at the pulmonary base alone* is *not very frequent There were only six examples in the 224 normal* cases of the present series But a systolic murmur *at the pulmonary base conducted down the left border of the sternum* to the apex is common There were 36 examples of this, or 16 per cent This pulmonary systolic murmur may be associated with an impulse which

gives to the examining hand almost the impression of a slight systolic thrill Radioscopy however reveals a perfectly normal heart with no increase in the size of the pulmonary conus This murmur is louder when the patient is lying down and may be present only in this position An apical systolic murmur is unlikely to be organic in origin if the pulmonary systolic part of the murmur is louder than that heard at the apex It may be stated that the possibility of organic disease diminishes in proportion to the number of these functional murmurs which are present

A systolic murmur which is heard *at the apex alone* is also common 25 such examples or about 11 per cent were present in this series The murmur was not conducted and frequently was absent when the patient was standing The murmur also often disappeared during full inspiration The third systolic murmur of importance was that heard at the apex and heard even more loudly towards the axilla during inspiration only There were 23 examples or 10 per cent in the series During expiration this murmur completely disappears This murmur is therefore called a *cardio insp ratory systolic murmur* It is common to find it in individuals who also show a systolic pulmonary murmur conducted to the apex No enlargement or radiological abnormality was found in these patients who frequently had a heart of the narrow longitudinal type quite obviously not organically diseased

Another common adventitious sound can be described as an *exocardial murmur or rub* There were 26 cases 11 per cent Whether this label exocardial has any basis in fact is impossible to determine One patient however produced some evidence which appeared to suggest that the sound arose outside the heart for there was a shuffling murmur which corresponded with all of the heart s movements systole and diastole of the ventricles and systole of the auricles so that a triple shuffling sound was present This is exactly analogous to what is frequently heard in pericarditis where the sound is definitely exocardial But in the present case the shuffling sound was completely eliminated by inspiration and was present only during the last half of expiration and the beginning of inspiration Moreover radiological examination showed the heart to be normal in size and shape and there were no symptoms what ever indicating any diminution in normal cardiac function This exceptional case is of importance only as presenting evidence for the exocardial basis of this type of murmur The commonest exocardial murmur is that which is heard most loudly in the fourth space to the left of the sternum and close to it The sound is

conducted to some extent towards the apex and to some extent towards the pulmonary base There is no associated thrill and no enlargement of the heart and in most cases the sound is eliminated during *full inspiration* *In about a quarter of the cases showing a* harsh exocardial sound this was present only at the pulmonary base Although a loud rather harsh shuffle is the usual character of the exocardial murmur in a few cases there was a definite musical squeak but this sound also went on full inspiration

These are the systolic murmurs of great importance which are found in normal young adults They suggest a variety of possible organic lesions and have to be differentiated from the murmur of mitral regurgitation which is present standing and lying although frequently it is louder in the latter position It is also not affected by respiration Furthermore in mitral regurgitation there is usually *some enlargement of the heart and the character of the first sound* may vary being raised in pitch The systolic murmurs at the pulmonary base are so common especially when conducted to the apex that they are unlikely to cause difficulty When the murmur is restricted to the pulmonary base some question of a minor organic congenital defect may arise Some of these pulmonary murmurs may be due to this cause but a diagnosis is impossible because the interference with the normal circulation if present is so slight as to cause neither symptoms nor signs There is no enlargement of the right ventricle cyanosis or clubbing The harsh exocardial murmurs to the left of the sternum suggest the possibility of chronic pericardial disease but here again the heart is otherwise absolutely normal and this diagnosis is therefore unlikely Another possibility is a very small patency in the interventricular septum But in this condition the murmur should be associated with a thrill and some enlargement of the heart and should be *audible during all phases of respiration*

The above murmurs and sounds have been fully described because a clear clinical description may be the first step in elucidating their method of causation Careful examination confirms the separate characters and qualities of these murmurs and by separating them from one another some impetus may be given to attempts to discover how each one may be produced In none of them has any characteristic abnormality been seen on the X ray screen Since the routine electrocardiogram was not taken in every case it is just possible that this form of investigation might be helpful although the examination of many thousands of normal people in hospital and *in private practice* has not provided evidence that this is likely to be the case However since these murmurs exist as clinical entities

it is necessary to define them in order that their innocence may be clearly established in individual patients

A final diagnostic difficulty which arises in apparently healthy young adults is that associated with the *blood pressure* This is frequently increased in nervous subjects and a figure of 180/90 mm of Hg may be observed Three points are of importance in determining whether this blood pressure increase is nervous or not The general behaviour of the individual is likely to give a clue to his nervous state An associated tachycardia is nearly always present when the blood pressure increase is due to nervous cause X ray measurement of the heart will show no increase whatever in size as would be the case if hypertension were constantly present Indeed the heart may be small The diastolic figure is far more important than the systolic If it is normal or only slightly raised the hyper tension is unlikely to have an organic cause

The most important points in assessing the state of the heart and cardiovascular system in young people are as follows

> Is there any enlargement of the heart? If the heart is normal in size and shape organic disease is unlikely Is the first sound of normal or of low pitch? If the latter is the case any associated *murmurs are not likely to be due to mitral disease Is the heart* small in size? Then organic disease is almost certainly absent Does the murmur disappear during respiration Any murmur which so disappears is very unlikely to be due to organic disease

During the examination of these 308 individuals the following abnormalities due to symptomless organic disease were noticed mitral stenosis was present in 43 cases mitral regurgitation in 13 mitral stenosis with aortic regurgitation in 4 aortic regurgitation almost certainly rheumatic was present alone in 7 patency of the ductus arteriosus was found in 2 and of the interventricular septum in 4 There was one example of transposition of the viscera and one of coarctation of the aorta In 9 the heart was enlarged X ray measurement showing cardiac diameter of more than half of the chest diameter The cause of this enlargement was not ascertainable

The organic defects observed in these cases were not accompanied by symptoms of any kind even when the young man or woman was playing normal strenuous games The chief difficulties in diagnosis of these organic lesions were as follows The murmurs of mitral disease were in some cases present only when the patient was lying down and in a few cases only in the left lateral position and after

exercise The diastolic murmur of aortic regurgitation was frequently so distant that it was difficult to detect and the leak was so small as to cause no blood pressure change The congenital murmurs were loud and easy to detect Nine cases of effort syndrome were present in the series In them the heart was organically normal but the symptoms were characteristic

CHAPTER XXXVI

CARDIAC SYMPTOMS AND SIGNS IN GENERAL *MEDICAL DISEASES*

CARDIAC symptoms and signs and general medical diseases may be related to one another in various ways. Cardiac patients may present a symptom or a sign which suggests disease primarily occurring in some other part of the body. For example cerebral disorders such as hemiplegia, may draw attention to the nervous system, pleurisy, cough and hæmoptysis may suggest a pulmonary abnormality, hæmaturia may draw attention to the renal tract, and liver tenderness and enlargement sometimes with jaundice, may divert the attention to the liver. A second group of diseases may be chiefly important to the cardiologist because they are extremely liable to injure the heart temporarily or permanently. For that reason a full knowledge of them is necessary to any physician especially interested in heart problems. Acute rheumatism, arteriosclerosis, syphilis and thyrotoxicosis are all examples. Finally a number of diseases generally regarded as being non cardiac in their manifestations, or in their effects not infrequently appear under a cardiac guise during the course of routine cardiological practice. It is with this group that the present discussion is chiefly concerned. The reason for possible confusion is that in many conditions cardiac symptoms and signs are found even in the absence of organic heart disease. Dyspnœa orthopnœa, lassitude, chest pain and palpitation are the symptoms most liable to draw attention towards the heart and away from the true cause, and œdema of the feet and legs, ascites, liver enlargement, and venous engorgement are the corresponding signs.

The first group of cases are examples in which *shortness of breath, undue fatigue*, and *palpitation* are especially prominent.

The first case was a man of 32, who for six months had been complaining of increasing dyspnœa and fatigue. At the time of examinatic
breath that
fatigue
for by smoking. No other symptoms were present. On examination

the heart was not enlarged clinically, and no abnormal heart signs or murmurs were detected The blood pressure was 110/70 The most striking physical sign was that in spite of his great shortness of breath there was no orthopnœa. The history gave a clue to the possible diagnosis, because his work was that of grinding steel objects upon a carborundum wheel in a motor-car factory X-ray examination confirmed that the heart was normal in size, but also showed advanced changes characteristic of silicosis

The second case was that of a barrister aged 60 who had remained in good health until ten weeks before the date of examination At that time he had an attack of what was called influenza, followed by "influenzal myocarditis" He had since then complained of persistent lassitude, some palpitation on exertion, and loss of appetite He had also lost thirteen pounds in weight He had always been extremely hard working and competent, and had been in the habit of smoking heavily and taking regularly a considerable quantity of alcohol, usually averaging half a bottle of whisky per day On examination the heart was normal The blood pressure was 138/80, and the heart rate was 90 A few fine râles were present at the left apex but these disappeared on coughing, and there were no other pulmonary abnormal physical signs The liver was enlarged three inches below the costal margin, and was firm and slightly irregular The electrocardiogram was normal X-ray screening revealed much old fibroid change, especially at the apex of the left lung, characteristic of chronic fibroid phthisis It was clear that pulmonary tuberculosis, superimposed upon cirrhosis of the liver, was the cause of his trouble

The third case was that of a woman of 28 Eighteen months previously she had been pregnant with her second child, and during the pr[illegible] appeared on the right side of the neck Th[illegible]

those of thyrotoxicosis The heart became rapid and irregular for a time after the operation She made a good recovery, and the pregnancy continued to term Ten months before the date of examination she had become pregnant again and a similar lump had appeared, together with tachycardia of 130, and also with exophthalmos and tremor In the sixth month of her pregnancy a partial thyroidectomy was done, together with a second removal of aberrant thyroid tissue Her thyrotoxic symptoms disappeared and the pregnancy continued to term The child was one month old when she was brought again for medical examination The heart rate was between 90 and 100

and she was complaining of fatigue, palpitation, and a little shortness of breath It had been suspected that thyrotoxicosis was once more present Physical examination showed pallor but not of a pronounced degree The heart was clinically normal, except for the first sound, which was slightly impure at the apex The blood pressure was 138/72 The electrocardiogram was normal The hæmoglobin however was only 54 per cent, and it was decided that her symptoms were due to an iron-deficiency anæmia She was given full doses of iron and made an uninterrupted and rapid recovery

The fourth case was that of a woman aged 40, who was examined in a small house at night at the request of her doctor The light in the room was not good, but it was clear that she was very ill and distressed, and very slightly jaundiced She was lying in bed on one pillow The heart rate was 120, and was regular On examination the heart was apparently normal except that the apex beat was in the mid-clavicular line But the stomach resonance was high, and the displacement of the apex beat was ascribed to some distension of the abdomen as a whole Tenderness was present over the abdomen The tenderness and discomfort were most marked over the left upper quadrant The history gave the clue to the probable diagnosis Three days previously she had fallen from household steps and her troubles started soon after this accident Enquiry as to the family history revealed that two sisters had suffered from slight jaundice all their lives, although the patient had not done so It was suspected that her present condition was due to a ruptured spleen and acholuric jaundice She was admitted to hospital, and exploration revealed a ruptured enlarged spleen hæmorrhage from which was the cause of her cardiovascular symptoms and signs She had acholuric jaundice

The most significant points from this group of cases can be summarised as follows The worker in the motor-car factory (case 1) had no orthopnœa, therefore no left heart failure was present He had no venous engorgement, therefore no right heart failure was present Finally the heart was not enlarged, therefore no myocardial disease was present In the case of the barrister (case 2), his lassitude was greater than his shortness of breath, and this relationship between these two symptoms is always a strong point against a cardiac cause In the case of the thyrotoxic woman (case 3), the anæmia was suspected by examination of the mucous membranes, for the colour of her cheeks, whether natural or acquired, was by no means pale and to that extent was deceptive Another point was that her skin was not warm and moist as would have been the case in thyrotoxicosis,

nor was it cold and moist as is found in effort syndrome Finally there was no tremor and she had had no loss of weight as would occur with an increased basal metabolic rate The woman with acholuric jaundice (case 4) was clearly very ill and shocked but in spite of the dyspnœa, which was in reality due to air hunger she was not orthopnœic and there was no great venous distension

The second group of cases is one in which *shortness of breath œdema of the legs and feet, and ascites* were the presenting clinical abnormalities The first case was that of a woman aged 37 who was lying in bed when first seen and whose complaints were that for the last few weeks she had been suffering from increasing shortness of breath and lassitude, and that recently ascites and some slight œdema of the legs had appeared On examination she was observed to be pale A systolic murmur was present loudest at the apex but also heard in the pulmonary area Slight pitting œdema was present in the legs and feet, and the abdomen was distended with fluid Her hæmoglobin was 50 per cent She was transferred to hospital and after paracentesis, a lump was palpated in the region of the stomach which proved to be a carcinoma

The second case was that of a man aged 59 whose complaint also was increasing tiredness, some swelling of the legs and feet and enlargement of the abdomen In addition the sight of his right eye had been less good within the last months On examination his hands were rather large and suggested the possibility of acromegaly, and for this reason his retinæ were examined carefully at once A black swelling looking like a lump of pitch was detected in the right retina The thoracic organs were normal A large irregular liver extending to the level of the umbilicus was palpated Some fluid was present in the abdomen and some swelling of the ankles was detected It was thought that he had a melanoma of the right eye, with secondary deposits in the liver This was confirmed when ferric chloride was added to the urine and a black deposit was precipitated

The third case was that of a man of 70, whose complaint also was increasing fatigue, enlargement of the abdomen with swelling of the legs and feet and some shortness of breath His thoracic organs were normal, but his abdomen was distended apparently with fluid Pitting œdema was present in the legs Catheterisation preparatory to paracentesis emptied an enormous bladder and this coincided with the disappearance of supposed ascites Further enquiries revealed the fact that he had been suffering from prostatic symptoms for a number of years

Considerations relative to this group of cases are as follows The woman with carcinoma (case 1) had both ascites and œdema of the legs, but the history revealed that the distension of the abdomen preceded the swelling of the legs Moreover the ascites was very considerable and the œdema of the legs was slight In heart failure the rule is, with a few exceptions, that œdema of the legs and feet precedes the occurrence of ascites by a definite period of time The exceptions to this rule are some cases of constrictive pericarditis and rare cases of mitral stenosis In the case of the man with melanotic sarcoma (case 2) it was the massive secondary deposits in the liver, which pressed upon the inferior vena cava, rather than increased pressure in the peritoneal cavity from fluid, which caused the swelling of the legs and feet

The third group of cases has especial reference to *chest pain* The first case was that of a man of 50, who was seen during the war in 1942 He was a busy business executive and was working eighty hours a week In addition to this he was doing "Home Guard" duties at the week-end Six weeks previously, while exercising in uniform on broken ground, he suffered a sudden onset of extreme dyspnœa with chest ache Since then shortness of breath had continued although not enough to immobilise him Also with the shortness of breath he had been conscious of an ache on the right side of his chest Except for a little cough at the start he had had no further symptoms Physical examination showed no cardiac abnormality The blood pressure was 116/78 The lungs were normal, except posteriorly at the right base, where distant rather cavernous breathing was heard No added sounds were present The electrocardiogram was normal, and on screening the chest a partial pneumothorax involving the right lower pleura was detected

The second case was that of a woman aged 46, whose chief complaint was pain of the classical angina of effort type, at first noticed only when going up hills, but latterly present on the level It was central, proportional to exercise, and radiated to the right arm She had to stand still after every twenty yards On examination the heart rate was 100 and the blood pressure 190/80 The apex beat was displaced to the left and there was a systolic murmur at the apex beat, conducted to the aortic base The electrocardiogram was normal The X-ray screening revealed general enlargement of the heart shape, involving the left ventricle The hæmoglobin was 29 per cent It was considered that she had hypertension with some coronary atheroma and a very severe secondary anæmia She was sent to bed and treated for an iron-deficiency anæmia, from which

she rapidly recovered Four months later she could walk without pain or any other symptoms, and her blood pressure had fallen to 158/92 She was seen eight years later and she was then in perfect health

The third case was that of a man aged 60, who also had pain of the angina of effort type In his case the cardiovascular system was found to be normal, and the cause was proved to be pernicious anæmia He was treated for this and was still well ten years later

The fourth case was that of a man of 65, whose chief complaint was severe attacks of pain across the centre of his chest during the previous two years Three months before he was examined he had an especially bad attack while in his office, since when he had not been feeling well Description of the pain revealed that it would last for something less than an hour, would increase during that time, and then tail off It was not accentuated by walking about, on the contrary this appeared to relieve the pain Profuse sweating occurred at the time, and he described the sensation as being that of a vice-like grip In between the attacks he was well On examination the heart rate was 90, the blood pressure was 158/96 A systolic murmur was present at the apex and conducted into the axilla and towards the aortic base The electrocardiogram was normal and X-ray measurement showed no enlargement of the heart It was decided that the attacks were not cardiac but were due to gall stone colic This was confirmed by cholecystogram

The fifth case was that of a man aged 50, a bus driver Six months previously, while driving his bus out of the garage and swinging it rapidly round the corner, he became giddy, pale somewhat collapsed, and vomited This seizure lasted ten to fifteen minutes He was taken to hospital, where he was kept in bed for six weeks with a diagnosis of heart block At the time of his sub sequent examination he explained that during his attack things went round and round," and that each time he looked up he felt giddy, and that this also occurred on moving the head from side to side There had been no real faintness or loss of consciousness He had never been ill in his life before, except for some persistent nasal catarrh Physical examination revealed a normal heart, the blood pressure was 156/90 The heart rate was 52 Inspection of previous electrocardiograms and that taken at the final examination showed a sinus bradycardia only, with a P–R interval of just under 0 2 of a second It was decided that there was no evidence of heart block, and that this had never been present, also that the attack

from which he had suffered was one of vestibular vertigo, secondary to nasal catarrh He was sent back to work

To recapitulate, in the course of cardiological practice the following variety of non-cardiac diseases was met with silicosis, fibroid pulmonary tuberculosis, cirrhosis of the liver, thyrotoxicosis, primary and secondary anæmias, acholuric jaundice, carcinoma of the stomach, melanomatosis, prostatitis, pneumothorax, cholecystitis, vestibular disease, and many others not recorded Great difficulties thus confront the individual who is attempting to acquire specialist knowledge in a medical subject like cardiology A specialist is apt to know more and more about less and less, and as applied to cardiology is apt to know more and more about the intricacies of cardioscopy or electrocardiography, and less and less about general medical diseases Since no individual can possibly retain a full knowledge of every branch of medicine, the would-be cardiologist must make up his mind as to his future path It is also clear that every cardiologist must have as good a general medical grounding as is humanly possible The ideal would appear to be close association of individuals in a team which should be composed of the pure scientist in this case the physiologist, the pure cardiologist whose function is rather that of advancing knowledge in special branches of cardiology, and finally the cardiologist with a knowledge of general medicine, or the general physician with special knowledge of cardiology There is much danger that subleties of this nature may not meet with the approval of individuals trying from office chairs to plan the future of medical services According to the predilection of such planners the importance of the various blends of cardiologist physician or physician cardiologist may not be given their full importance The only safeguard would appear to be to allow individual physicians to choose their own niche, according to their personal aptitudes Having chosen their individual paths they should be encouraged to fit into teams according to their personal powers and desires

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